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CONTENTS

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IN accordance with the requirements of the Farm Relief Act and the National Industrial Recovery Act, the virus-serum industry as a whole has formulated a code of fair competition and marketing agreement which has been submitted to the Secretary of Agriculture at Washington for his approval.

THE problems of adequate wages and maximum hours of labor, of fair and unfair trade practices, of uniform prices to various classes of purchasers has been considered in the adopted code.

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Vol. LXXXIII, N. S. Vol. 37 SEPTEMBER, 1933 No. 3

THE CHICAGO MEETING

The 1933 convention is now a part of A. V. M. A. history. Without any question, this meeting, the fourth held in Chicago in a span of 43 years, will take rank among the big conventions. There are those who predicted all sorts of attendance figures for the meeting this year, even as high as twice the best previous number, but when the registration desk suspended operations late on Friday afternoon the attendance figure was just shy of the 1,400 mark. An examination of the registration list, after the close of the meeting, showed that quite a few who were in attendance apparently had failed to register. This is always the case and probably always will be. So, from the standpoint of attendance, the 1933 meeting compares favorably with Kansas City, in 1931; Detroit, in 1929, and Minneapolis, in 1928.

There were four business sessions. These were marked by the dispatch with which the routine business of the Association was transacted. This meeting really marked the end of the custom of having reports of all committees and officers received by the membership at large, in view of the fact that final approval was placed on a number of amendments to the Constitution and By-Laws, which, in effect, delegate this function to a new body, the House of Representatives. For a number of years it has

required three or four half-day sessions to transact business. Under the new plan, this will be handled by the House of Representatives, and it should not require more than one half-day session for the Association to handle all of the strictly business matters to come before it. The net result of the change should be two, or even three, half-day sessions that may be devoted to the reading and discussion of scientific papers.

The address of President Williams, published in full in this issue of the JOURNAL, was a scholarly masterpiece. It clearly defined the place which veterinary science has occupied, from Biblical times down to the present. It warned members of the veterinary profession that they should put and keep their own house in order. It delivered a stinging rebuke to those agencies that have tended to undermine the field of the practitioner. In its entirety, the address showed an unusual grasp of the present situation as it affects the veterinarian individually and the profession as a whole.

The outstanding committee report was that of the Special Committee on Agricultural Extension Service, delivered to a crowded house on Thursday afternoon. There was not a vacant chair in the room when Dr. H. E. Curry finished the reading of the report, and the undivided attention given the speaker by the largest audience of the week gave testimony to the importance of the subject, as viewed by those who have kept in touch with the situation. Further comment will not be made here, as the report in full will be published in the JOURNAL next month.

The election of officers injected considerable interest into the meeting. For the second time during recent years, it became necessary to resort to the ballot-box. Something that was appreciated by but very few was the fact that printed ballots, bearing the names of all candidates for the contested offices, were available to the electorate, when the polls were opened at 9 o'clock on Tuesday morning, for the first time in A. V. M. A. history. On all previous occasions it has been necessary for each voter to write in the names of those for whom he wished to vote. The Election Committee worked steadily from 9 o'clock in the morning until after the same hour that night supervising the issuance and casting of the ballots and the counting and tabulating of the votes. The election was systematic and orderly at all times, and many comments were heard concerning the improvement over the old system that was in vogue for so many years.

There were 407 ballots cast and the official canvass disclosed that Dr. C. P. Fitch, of Minnesota, had won over Dr. J. S. Koen,

of Missouri, for the office of President. When the official report of the election was given to the Association, at the Wednesday afternoon session, Dr. Koen very graciously moved that the election of Dr. Fitch be made unanimous. This occasion marked the third time in recent years that a member of the Executive Board has been elevated to the presidency. In 1928, Dr. T. E. Munce was so honored; in 1932, Dr. N. F. Williams was advanced in like maner.

The five vice-presidents selected are as follows:

First Vice-President: Dr. W. E. Cotton, Bethesda, Md.

Second Vice-President: Dr. G. A. Dick, Philadelphia, Pa.

Third Vice-President: Dr. W. G. Hollingworth, Utica, N. Y.

Fourth Vice-President: Dr. S. W. Haigler, St. Louis, Mo.

Fifth Vice-President: Major Harry E. Van Tuyl, V. C., U. S. Army.

The selection of the vice-presidents exemplified the generosity, liberality, or whatever one chooses to call it, of the A. V. M. A. members in distributing these honors. Five distinct branches of the veterinary profession were recognized: Dr. Cotton, of the U. S. Bureau of Animal Industry, represents research; Dr. Dick, of the University of Pennsylvania, the colleges; Dr. Hollingworth, of the Utica (N. Y.) Department of Public Safety, public health; Dr. S. W. Haigler, the practitioners, and Major Van Tuyl, the Army.

Dr. M. Jacob, of Knoxville, Tenn., was reëlected treasurer for his sixteenth consecutive term. The Executive Board reëlected Dr. Robert S. MacKellar, of New York City, to the chairmanship for his fourth consecutive term.

The symposium on Bang's disease, held as a general session on Tuesday morning, really was a new departure. It gave a suggestion of what may be expected in the form of general sessions in the future, when the House of Representatives is functioning and handling the routine business of general sessions of the past. We can visualize a program with general sessions devoted entirely to papers and discussions dealing with important subjects, when everybody may attend without having to miss other parts of the program.

The sectional meetings held Wednesday and Thursday mornings were well attended. However, the two practice sections drew the bulk of the attendance. After all, general practice engages the attention of the largest number of veterinarians and this probably will continue to be the case for many years to come. The program of any strictly veterinary meeting that does not

take this fact into account is very apt to fall short of fulfilling its purpose.

The alumni luncheons on Wednesday taxed even the facilities of the Palmer House, where more meals are served, under one roof, every day in the year, than in any other place in the world. Up until about an hour before the scheduled time for the luncheons, about 160 tickets had been sold. During the hour just before the luncheons, over 300 additional tickets were sold to those who suddenly decided that they wanted to attend. As a result, 470 were served, with very little delay, in thirteen different groups: Chicago, Colorado, Cornell, Iowa, Kansas City, Kansas State, McKillip, Michigan, New York University, Ohio, Ontario, Pennsylvania and Washington.

The banquet served to about 450 Wednesday evening was a colorful event. The entertainment features were somewhat out of the ordinary and everybody seemed to enjoy them, particularly those in the bald-headed row. Here several of the highbrows were much impressed, or imprinted, or "sumpin," by Miss Doris Hurtig, when she bestowed her favors among the audience. The Chinese Ambassador and the Careless Waiter created lots of amusement, so that everybody was in a jolly mood by the time that the serious part of the program was reached.

In the role of toastmaster, Dr. H. D. Bergman added to his reputation as a master of ceremonies. To say that he was in his usual good form is putting it entirely too mildly. He first presented Miss Marion Jaffray, talented daughter of one of Chicago's prominent practitioners. She rendered two numbers at the piano that simply brought down the house. The first speaker of the evening was Mr. W. S. Corsa, of Whitehall, Illinois, whose topic was "Our Animal Industry." Mr. Corsa indicated a keen appreciation of the value of an adequately trained veterinary personnel to the welfare of agriculture in general and animal husbandry in particular.

Then came another outstanding feature of the meeting, in the award of gold medals to Drs. Cooper Curtice and Fred L. Kilborne, of southern cattle fever fame. Dr. Curtice was present to receive his medal, but Dr. Kilborne, on account of poor health, could not be in attendance, and Dr. Bergman awarded his medal *in absentia*. It will be delivered to Dr. Kilborne by Dr. W. A. Hagan. Thus was kept in motion a custom that was started at Atlanta, one year ago, when gold medals were presented to four members of the A. V. M. A. who had been on the roll fifty years or more. The veterinary profession has been backward in honor-

ing those who have contributed distinguished services to the cause of veterinary medicine, particularly in its relation to public health.

The second speaker was an honorary member of the A. V. M. A., Dr. W. A. Evans, Health Editor of the Chicago *Tribune*, who spoke on "Taking Health to the People." Dr. Evans is in the habit of addressing all kinds of audiences and his versatility was plainly in evidence on this occasion, when he interestingly reviewed some of the experiences in his career as physician, health commissioner and editor. Following the banquet, all adjourned to the Red Lacquer Room for the President's reception and dance. The arrival of the midnight hour brought to a close one of the most brilliant affairs in A. V. M. A. convention history.

The clinic on Friday was held right in the spacious Exhibition Hall of the Palmer House, as previously arranged. The animals, both large and small, were there, as promised. The poultry clinic, consisting of over 200 chickens and other varieties of fowl, the contribution of the University of Illinois, under the direction of Dr. Robert Graham, was complete to the last detail. Until late in the afternoon, clinics for both large and small animals were kept going. In fact there was so much program material that an overflow meeting had to be arranged in the Grand Ballroom in the afternoon. Easily the feature of the clinic for small animals was the demonstration of classical surgical technic by Dr. Frank C. Mann, of the Mayo Foundation, Rochester, Minnesota.

The festivities of the week were brought to a close Friday evening, with a delightful boat ride on Lake Michigan, enjoyed by almost 500 members and visitors. Here was given an opportunity to see Chicago's lake-front at night and the brilliant illumination of A Century of Progress. When the S. S. *Florida* tied up at the Navy Pier just before midnight, there were about 500 tired people who realized that they had just about reached the end of a rather strenuous week in the Windy City. The combination of A Century of Progress and the 70th annual convention of the American Veterinary Medical Association will be hard to beat.

And now for New York in 1934 and the Twelfth International Veterinary Congress!

National Dog Week—October 1-7, 1933.
A Pedigreed Dog in Every Home.

CONVENTION NOTES

Dr. L. G. Combs, of Balboa, was the single registrant from the Canal Zone.

All the way from Hawaii came Drs. Lloyd C. Moss and Ernest H. Willers, of Honolulu.

Dr. Francisco Moguel, of Nuevo Laredo, Tamps., Mexico, enjoyed himself renewing acquaintances he had made on previous visits to Chicago.

The meeting took on an international appearance with veterinarians present from Canada and Mexico. Hawaii and the Canal Zone also had members present.

North Carolina came through with five registrations: Drs. P. M. Abernethy, Burlington; L. J. Faulhaber, A. A. Husman and William Moore, Raleigh; M. M. Leonard, Asheville.

Five *West Virginians* were among those present: They were: Drs. John L. Core, Shinnston; R. C. Dickson, S. E. Hershey and H. M. Newton, Charleston. H. B. Langdon, Charles Town.

Maine did well to have five representatives present. They were: Drs. J. R. Corliss, Augusta; C. F. Davis, Rumford; R. E. Libby, Richmond; E. E. Russell, Farmington; C. L. Ryan, Dexter.

Seven tickets from *Georgia*: Drs. Hugh F. Arundel, Quitman; C. H. Ellis, Jr., Brunswick; J. L. Hopping, L. J. Kepp and J. W. Thome, Atlanta; J. L. Miller, Columbus; J. M. Sutton, Sylvester.

Another distant state with five in attendance was *Oregon*. Drs. E. M. Dickinson, O. H. Muth and B. T. Simms registered from Corvallis; Dr. James B. Harrison, from Portland; Dr. W. H. Lytle, from Salem.

Just a half-dozen registered from *Louisiana*: Drs. H. H. Baur, Monroe: E. P. Flower and C. M. Heflin, Baton Rouge; K. W. Franks, Minden; Louis Leonpacher, Lafayette; Hamlet Moore, New Orleans.

From *Maryland* there were six members on the registry: Drs. W. E. Cotton, Bethesda; R. W. Culbert and E. B. Dibbell, Baltimore; George A. H. Edminston, Easton; J. W. Hughes, Elkton; Mark Welsh, College Park.

There were seven registered from *Colorado*: Drs. A. A. Hermann, J. N. Huff, G. G. Miller and G. H. Oliver, Denver; I. E. Newsom, Fort Collins; M. E. Spratlin, Littleton; Charles C. Stewart, Colorado Springs.

Only five states were not represented at the meeting: Arizona, Idaho, Nevada, New Mexico and Vermont.

Of the three living ex-treasurers of the A. V. M. A., Dr. F. H. Schneider, of Philadelphia, was the only one present. Dr. William Herbert Lowe, of Paterson, N. J., who served from 1897 until 1905, was seriously ill and wired his regrets.

Every member of the A. V. M. A. who served as secretary since 1910 was in attendance. The list includes: Drs. C. J. Marshall (1910-13), N. S. Mayo (1913-15 and 1918-22), C. M. Haring (1915-16), L. A. Merillat (1916-17), L. E. Day (1917-18) and H. Preston Hoskins (1922-).

An octet of *Massachusetts* members enjoyed the meeting. They were: Drs. Walter R. Carroll, Brookline; W. H. Dodge, Leominster; Charles S. Gibbs, Amherst; Harry W. Jakeman, Boston; William F. McNamara and Warren L. Thayer, Worcester; G. F. O'Malley, Clinton; L. A. Paquin, Webster.

Three states, Montana, Rhode Island and Washington, had one member each on the registry. Dr. Frank M. Nelson, of Livingston, represented *Montana*. Dr. Thomas E. Robinson, of Westerly, was *Rhode Island's* delegate, and Dr. E. E. Wegner, of Pullman, was the member from *Washington*.

An even dozen ex-presidents of the A. V. M. A. attended the meeting. They were: Drs. J. R. Mohler (1912-13), C. J. Marshall (1913-15), C. E. Cotton (1916-17), C. A. Cary (1919-20), A. T. Kinsley (1921-22), W. H. Welch (1922-23), C. H. Stange (1923-24), L. A. Merillat (1924-25), T. A. Sigler (1926-27), T. E. Munce (1928-29), T. H. Ferguson (1929-30) and R. R. Dykstra (1931-32).

Nine of the veterinary colleges were represented by their respective deans: Cary, of Alabama Polytechnic Institute; Brumley, of Ohio State University; Dick, of the University of Pennsylvania; Dykstra, of Kansas State College; Giltner, of Michigan State College; Hagan, of Cornell University; McGilvray, of Ontario Veterinary College; Stange, of Iowa State College, and Wegner, of the State College of Washington.

Each of the Dakotas had seven members at the meeting. From *North Dakota*: Drs. B. K. Bjornson, Mandan; T. O. Brandenburg, Bismarck; B. W. Coons, Lisbon; J. H. Ehlers, Pembina; O. D. Foss, Christine; Lee M. Roderick, Fargo; S. S. Westgate, Grafton. From *South Dakota*: Drs. E. L. Eggleston, Alcester; C. H. Hays, Pierre; T. H. Kindred, W. R. Laird and M. W. Ray, Sioux Falls; L. S. Leider, Howard; G. S. Weaver, Brookings.

The Keystone State was forward in the line with thirty-two in attendance. The *Pennsylvania* list included: Drs. E. P. Althouse, Sunbury; M. F. Barnes, Harry W. Herriott, and T. E. Munce, Harrisburg; A. F. Baver, Kutztown; Clarence E. Bley, Chester; James E. Burton, Slippery Rock; C. M. Christy, Brookville; M. A. Davies, Troy; Thomas Castor, G. A. Dick, Major Henry E. Hess, Drs. William H. Ivens, C. J. Marshall and F. H. Schneider, Philadelphia; C. D. Evans, Kane; B. B. Farr, Meshoppen; C. W. Frush, Samuel Glenn, Pittsburgh; A. C. Galbraith, Newville; R. L. Kann, Mechanicsburg; G. M. Leighow, Danville; S. M. Nissley, Bellefonte; T. F. Nugent, New Castle; H. B. Prothero, Johnstown; John Reichel, Glenolden; M. D. Rentschler, Punxsutawney; H. B. Roshon, Reading; E. L. Simpson, Westfield; H. E. Skoog, Pittston; H. S. Stoker, Stillwater; J. H. Turner, Wellsboro.

Minnesota had a sizable representation of thirty-nine: Drs. Earl H. Allinson, Balaton; A. M. Anderson, Luverne; W. L. Boyd, Miles L. Davenport, C. R. Donham, Leo S. Englerth, R. Fenstermacher, C. P. Fitch, H. C. H. Kernkamp, B. A. Pomeroy and M. S. Whitcomb, Saint Paul; E. L. Carpenter, Minnesota Lake; John Enama, Lake City; C. B. Erickson, Pelican Rapids; J. J. Findlay, Duluth; I. M. Ford, Edgerton; J. P. Foster, D. M. McDonald and O. B. Morgan, Minneapolis; J. R. Friedline, Wood Lake; Carl Hansen, Faribault; E. H. Kartrude, Jasper; Harry L. Kidd, Grand Meadow; John W. Kummer, Hastings; T. M. Lee, Watertown; William McLaughlin, Rush City; R. A. Merrill, Clara City; C. A. Nelson, Brainerd; Carl Olson, Jr., and C. F. Schlotthauer, Rochester; D. R. Philp, Mankato; L. H. Phipps, Winnebago; M. H. Rolighed, Appleton; R. Schaap, Pipestone; Edward E. Thompson, Lyle; J. E. Thompson, Ely; Ralph L. West, Waseca; C. H. Wetter, Princeton; W. H. Whitcomb, Plainview.

Missouri sent forty-two progressive veterinarians: Drs. S. B. Aldridge, Richmond; James A. Austin, E. A. Cahill, J. C. Flynn, Charles D. Folse, A. T. Kinsley, Ashe Lockhart, C. M. McFarland and C. E. Salsbery, Kansas City; George E. Bartholomees, Sheldon; William E. Beckmann, C. L. Campbell, Milton R. Fisher, S. W. Haigler, J. T. Jennemann, Ernest C. McCulloch, J. S. Koen, W. A. Parker and H. B. Wood, Saint Louis; J. L. Cherry, Tarkio; H. C. Conrad, Polo; G. K. Clark, Hannibal; J. W. Connoway, O. S. Crisler, A. J. Durant, Cecil Elder, A. M. McCapes and Stanley Smith, Columbia; Hugh E. Curry, Jefferson City; W. C. Dillard, Farmington; A. Goodlive, Marshall; G. R. Killian, Web-

ster Groves; Major Paul R. King, Jefferson Barracks; Drs. J. B. Latshaw, Caruthersville; W. E. Martin, Perry; J. S. Matteson, Parnell; C. D. Meredith and W. J. Stone, Joplin; R. P. Poage, Shelbina; E. A. Shikles and J. R. Shikles, Dearborn; A. A. Taylor, Mendon.

New York came forward with forty-three from the Empire State: Drs. E. B. Ackerman, Huntington Bay Hills; F. W. Andrews, Mount Kisco; F. J. Baker, Gouverneur; H. W. Bales, Oswego; M. D. Baum, R. E. Geisler, John Jackson, Robert S. MacKellar, John J. Regan, C. G. Rohrer and C. P. Zepp, New York City; R. R. Birch, E. L. Brunett, H. H. Dukes, J. N. Frost, W. A. Hagan, Seth D. Johnson, H. J. Milks, H. C. Stephenson, Earl Sunderville and A. Zeissig, Ithaca; Don A. Boardman, Rome; Frank J. Brands and H. Kock, Brooklyn; J. Elliott Crawford, Far Rockaway; C. E. De Camp, Nepera Park; W. A. Dennis, Jamestown; David F. Deming, Massena; J. F. Devine, Goshen; F. F. Fehr, Buffalo; E. T. Faulder, William Henry Kelly, J. G. Wills, Albany; W. G. Hollingworth, Utica; David M. Hoyt, Canastota; H. K. Leonard, Mexico; B. M. Lyon and Norman J. Pyle, Pearl River; H. K. Miller, Mamaroneck; Shefford S. Miller, New Rochelle; A. J. Tuxill, Auburn; Bert R. Wilbur, South Dayton; Millard M. Woods, Jackson Heights.

Indiana was well represented with fifty-nine in attendance: Drs. W. J. Armour and C. C. Winegardner, Goshen; J. L. Axby, Lawrenceburg; D. D. Baker, Wabash; G. W. Bardens, Lowell; C. R. Baumgartner and G. E. Botkin, Marion; S. E. Bowman, Odon; F. W. Bratten, McCordsville; F. E. Broad, Plymouth; Frank H. Brown, H. Busman, C. C. Donelson, R. C. Julien, Frank J. Muecke, J. C. Schoenlaub and J. D. Stillwell, Indianapolis; C. E. Chapin, Fremont; G. L. Clark, Columbia City; R. A. Craig and L. P. Doyle, West Lafayette; J. B. Current, Topeka; O. B. Curry, Morristown; E. H. Daley, La Porte; H. W. Demsey, Huntington; Harry W. Brown, G. M. Dorman, George W. Gillie, Charles J. Gruber and Paul C. Kucher, Fort Wayne; Glenn L. Ebright, Hammond; Lawrence J. Etnire, Williamsport; A. F. Ferguson. LaFontaine; L. C. Finley, Lapel; Walker France, Boonville; D. W. Gerber, Clay City; George D. Haimbaugh, Rochester; D. C. Hancock, Mays; O. W. Hiner, Butler; C. J. Hufty, Burlington; R. Hyde, Hartford City; Thomas F. Hyde, Brookville; W. C. Kortenber, New Haven; W. H. Lane, Camden; H. J. Magrane, Mishawaka; W. B. Massie, Boston; J. H. Mills, Russiaville; G. W. Musselman, Denver; Vernon B. Overman, Lynn; O. C. Shockley, New Ross; Payson Schwin, Elkhart; C. F.

Shartle, Stilesville; T. A. Sigler, Greencastle; C. Harvey Smith, Crown Point; G. O. Smith, Ligonier; F. C. Tucker, Claypool; H. E. Whiffing, Lebanon; Roy B. Whitesell, Lafayette; R. E. Wood, Rockville.

Iowa's delegation of seventy was the second largest at the convention. Present from this state were: Drs. N. W. Ackerman, Van Horne; L. C. Ball, Titonka; C. H. Banks, Tipton; J. A. Barger, Howard T. Corbet, J. I. Gibson, A. H. Quin and H. A. Seidell, Des Moines; Major Jesse D. Derrick, Fort Des Moines; Drs. John Doerr, Melbourne; R. E. Elson, Vinton; G. B. Fincham, G. P. Statter, Sioux City; E. A. Benbrook, H. D. Bergman, H. L. Foust, E. A. Hewitt, C. D. Lee, C. N. McBryde, L. W. McElyea, W. C. Merchant, N. L. Nelson, F. D. Patterson, L. H. Schwarte, C. H. Stange, F. E. Walsh, T. W. Workman and Major H. J. Juzek, Ames; Drs. Robert K. Benn, Packwood; R. C. Byrnes, Traer; Thomas W. Gidley, Malvern; J. A. Given, Marcus; C. J. Heckard, Wheatland; Henry Hell, Wilton Junction; H. J. Hoffeins, Denison; Kress Johnson, Prairie City; L. W. Kellogg, Anamosa; N. A. Kippen, Independence; Frank C. Kochendorfer, Decorah; H. C. Kreuger, Mason City; A. H. Kraus, Marengo; H. E. Leach, Avoca; J. A. Lueth and T. A. Shipley, Council Bluffs; J. H. McLeod, Charles City; Will F. Miller, Storm Lake; A. R. Menary and Grant B. Munger, Cedar Rapids; A. J. Miller, Granville; W. S. O'Brien, Ryan; M. W. Radloff, Le Grand; L. E. Ragan, Mitchellville; W. B. Redman, Dow City; L. P. Scott, Waterloo; C. J. Scott, Knoxville; D. E. Baughman, H. J. Shore and H. C. Smith, Fort Dodge; J. H. Spence, Clinton; H. L. Stewart, Chariton; J. J. Strandberg, Belle Plaine; R. H. Tesdell, Ogden; L. A. Tischhauser, Garnavillo; W. C. Vollstedt, Dixon; R. L. Ward, Batavia; F. D. Weimer, Cumberland; C. W. Wiley, Farson; F. M. Wilson, Mechanicsville; E. L. Wolff, Avoca; Laird Woods, Malcom.

APPLICATIONS FOR MEMBERSHIP

(See July, 1933, JOURNAL)

FIRST LISTING

BETZOLD, CURTIS W. 4907 King Hill Ave., Saint Joseph, Mo.
D. V. M., Cornell University, 1932
Vouchers: William E. Jennings and B. J. Stockler.

BRAMER, CLARENCE N. 1817 Church St., Evanston, Ill.
D. V. M., Cornell University, 1923
Vouchers: J. V. Lacroix and E. C. Khuen.

BRATT, H. M. 706 S. 24th St., Terre Haute, Ind.
D. V. M., Terre Haute Veterinary College, 1915
Vouchers: C. C. Donelson and L. A. Merillat.

BURNHAM, SCOTT A. 1640 Linden, Oklahoma City, Okla.
D. V. M., Colorado Agricultural College, 1925
Vouchers: C. C. Hisel and L. J. Allen.

CLARK, GEO. L. 110 Jackson St., Columbia City, Ind.
V. S., Ontario Veterinary College, 1905
Vouchers: F. W. Bratten and A. F. Ferguson.

DUNHAM, T. F. Clinton, Okla.
D. V. M., Kansas City Veterinary College, 1913
Vouchers: C. C. Hisel and Fred S. Molt.

ETCHISON, ARCHIE C. Assumption, Ill.
M. D. C., Chicago Veterinary College, 1910
Vouchers: C. L. Campbell and W. H. Welch.

FISHER, MILTON R. 9 Municipal Bldg., Saint Louis, Mo.
D. V. M., Ohio State University, 1925
Vouchers: J. S. Koen and Ernest McCulloch.

FRANCE, WALKER Boonville, Ind.
D. V. M., Terre Haute Veterinary College, 1913
Vouchers: Frank H. Brown and L. A. Merillat.

GLENN, H. H. 615 N. E. 7th St., Oklahoma City, Okla.
M. D. C., Chicago Veterinary College, 1902.
Vouchers: L. J. Allen and C. C. Hisel.

GRACE, MAJ. CHARLES O. Fort Benning, Ga.
D. V. M., Cornell University, 1917
Vouchers: Maj. Seth C. Dildine and Maj. F. L. Holycross.

GULICK, G. G. 2725 N. W. 21st St., Oklahoma City, Okla.
D. V. M., Kansas City Veterinary College, 1916
Vouchers: C. C. Hisel and L. J. Allen.

HAYER, O. T. Carthage, Ill.
D. V. M., Chicago Veterinary College, 1915
Vouchers: J. S. Koen and H. D. Chamberlain.

HILL, GEO. H. 611 N. Aldrich St., Geneseo, Ill.
M. D. C., Chicago Veterinary College, 1911
Vouchers: C. W. McLaughlin and H. W. Jakeman.

HORSLAND, JAMES E. c/o P. Burns & Co., Ltd., Calgary, Alta., Can.
B. V. Sc., Ontario Veterinary College, 1931
Vouchers: R. T. Skelton and R. C. Duthie.

JONES, T. A. 614 N. Central, Oklahoma City, Okla.
D. V. S., Kansas City Veterinary College, 1904
Vouchers: C. C. Hisel and L. J. Allen.

KINCAID, ALBERT R. Stonington, Ill.
D. V. M., Chicago Veterinary College, 1914
Vouchers: E. L. Quitman and J. V. Lacroix.

KITZHOFER, JOSEPH H. 423 N. W. 32nd St., Oklahoma City, Okla.
D. V. M., Ohio State University, 1910
Vouchers: C. C. Hisel and L. J. Allen.

LANG, J. P. Crystal Lake, Ill.
M. D. C., Chicago Veterinary College, 1907
Vouchers: Herman C. Rinehart and W. B. Holmes.

LEHMAN, J. L. Edgar, Wis.
D. V. M., McKillip Veterinary College, 1914
Vouchers: John R. Berggren and C. M. Heth.

LEONHART, O. H. 1621 Birch, Oklahoma City, Okla.
M. D. C., Chicago Veterinary College, 1900
Vouchers: L. J. Allen and C. C. Hisel.

MCCOY, J. A. Washington Court House, Ohio
D. V. M., Ohio State University, 1911.
Vouchers: J. W. Jackman and E. L. Roshon.

MASSIE, W. B. Boston, Ind.
D. V. M., Michigan Agricultural College, 1916
Vouchers: J. H. Mills and C. J. Hufty.

MEREDITH, S. M. 1410½ N. W. 21st St., Oklahoma City, Okla.
D. V. S., Kansas City Veterinary College, 1906
Vouchers: C. C. Hisel and L. J. Allen.

MOGUEL M., FRANCISCO
Gral. Gutierrez 1708, Nuevo Laredo, Tamps., Mexico
....., University of Mexico, ...
Vouchers: N. F. Williams and John R. Mohler.

O'NEAL, C. E. Box 725, Jackson, Miss.
D. V. M., Kansas State College, 1916.
Vouchers: R. H. Stewart and E. H. Durr.

PARKER, ROY R. 3317 W. 17th St., Oklahoma City, Okla.
D. V. M., Kansas State College, 1919
Vouchers: L. J. Allen and W. L. Hiatt.

SCHAFSTALL, ALBERT C. New Washington, Ohio
B. V. Sc., Ontario Veterinary College, 1910
Vouchers: Clinton D. Barrett and Harry A. Hoopes.

SCHWERMANN, H. E. New Ulm, Minn.
D. V. M., Iowa State College, 1931
Vouchers: C. D. Lee and Carl Olson, Jr.

SEIDELL, H. A. 710 Prospect Rd., Des Moines, Iowa
D. V. M., Iowa State College, 1917
Vouchers: J. A. Barger and H. J. Shore.

STOKER, H. S. Stillwater, Pa.
V. M. D., University of Pennsylvania, 1903
Vouchers: Edward P. Althouse and Martin L. Hutchins.

STRANGE, C. ROY State Capitol, Madison, Wis.
D. V. M., Colorado Agricultural College, 1923
Vouchers: W. Wisnicky and V. S. Larson.

STULTS, JOSEPH W. 629 N. E. 11th St., Oklahoma City, Okla.
M. D. V., McKillip Veterinary College, 1911
Vouchers: C. C. Hisel and L. J. Allen.

TURK, R. D. Kansas State College, Manhattan, Kan.
D. V. M., Kansas State College, 1933
Vouchers: R. R. Dykstra and E. E. Leasure.

VILOTT, W. T. 909 N. E. 29th St., Oklahoma City, Okla.
D. V. M., Kansas City Veterinary College, 1912
Vouchers: L. J. Allen and W. L. Hiatt.

WADLEIGH, HERBERT C. Seaton, Ill.
D. V. M., Colorado Agricultural College, 1920
Vouchers: C. W. McLaughlin and Grant B. Munger.

WALSH, F. E. Iowa State College, Ames, Iowa
D. V. M., Iowa State College, 1918
Vouchers: C. D. Lee and F. D. Patterson.

Applications Pending

SECOND LISTING

(See August, 1933, JOURNAL)

Branigan, Chester P., 10759 Washtenaw Ave., Chicago, Ill.
Case, Ralph W., Box 1286, Prescott, Ariz.
Collins, George L., Stanton, Neb.
Cox, Norman H., 264 S. Main St., Rutland, Vt.
Craver, Thomas W., 234 5th Ave., Youngstown, Ohio.
Granholm, Paul R., 605 N. 13th St., Milwaukee, Wis.
Kight, A. B., 324 S. Kimball St., Casper, Wyo.
Lenheim, Edward H., 326 City Building, Topeka, Kan.
Munn, Urton, Tallassee, Ala.
Tennille, Newton B., Wapakoneta, Ohio.
Wolf, Herman H., 1713 W. Slauson Ave., Los Angeles, Calif.

The amount which should accompany an application filed this month is $6.67, which covers membership fee and dues to January 1, 1934, including subscription to the JOURNAL.

COMING VETERINARY MEETINGS

New York City, Veterinary Medical Association of. Academy of Medicine, 5th Ave. and 103rd St., New York, N. Y. September 6, 1933. Dr. John E. Crawford, Secretary, 708 Beach 19th St., Far Rockaway, Long Island, N. Y.

East Tennessee Veterinary Medical Society. White Surgical Supply Co., Knoxville, Tenn. September 9, 1933. Dr. R. E. Baker, Secretary, Morristown, Tenn.

Interstate Veterinary Medical Association. Elks Building, Omaha, Neb. September 11, 1933. Dr. G. L. Taylor, Secretary, Plattsmouth, Neb.

Chicago Veterinary Medical Society. Hotel LaSalle, Chicago, Ill. September 12, 1933. Dr. O. Norling-Christensen, Secretary, 1904 W. North Ave., Chicago, Ill.

Southeastern Michigan Veterinary Medical Association. Detroit, Mich. September 13, 1933. Dr. A. S. Schlingman, Secretary, Parke, Davis & Co., Detroit, Mich.

Tulsa County Veterinary Association. Tulsa, Okla. September 14, 1933. Dr. J. M. Higgins, Secretary, 3305 E. 11th St., Tulsa, Okla.

National Veterinary Medical Association Congress. Town Hall, Llandudno, Wales. September 18-23, 1933. Mr. F. Knight, General Secretary, 2, Verulam Buildings, Gray's Inn, London, W. C. 1, England.

Kansas City Veterinary Association. Baltimore Hotel, Kansas City, Mo. September 19, 1933. Dr. J. D. Ray, Secretary, 1103 E. 47th St., Kansas City, Mo.

Southern California Veterinary Medical Association. Chamber of Commerce Building, Los Angeles, Calif. September 20, 1933. Dr. T. G. Beard, Secretary, 3684 Beverly Blvd., Los Angeles, Calif.

American Public Health Association. Indianapolis, Ind. October 9-12, 1933. Willimina Rayne Walsh, Secretary, 450 Seventh Ave., New York, N. Y.

Eastern Iowa Veterinary Association, Hotel Montrose, Cedar Rapids, Iowa. October 10-11, 1933. Dr. Iva Dunn, Secretary, Atkins, Iowa.

American Humane Association. Hartford, Conn. October 10-12, 1933. Mr. N. J. Walker, General Manager, 80 Howard St., Albany, New York.

Maine Veterinary Medical Association. State House, Augusta, Me. October 11, 1933. Dr. L. E. Maddocks, Secretary, R. F. D. 2, Augusta, Me.

Inter-State Veterinary Medical Association. Sioux City, Iowa. October 12-13, 1933. Dr. W. A. Aitken, Secretary, Merrill, Iowa.

International Association of Dairy and Milk Inspectors. Hotel Claypoole, Indianapolis, Ind. October 12-14, 1933. Dr. Paul B. Brooks, Secretary, Deputy Commissioner of Health, New York State Department of Health, Albany, N. Y.

Illinois Veterinary Conference, University of. University of Illinois Medical School, Chicago, Ill. October 16-18, 1933. Dr. Robert Graham, Secretary, University of Illinois, Urbana, Ill.

Keystone Veterinary Medical Association. Philadelphia, Pa. October 25, 1933. Dr. C. S. Rockwell, Secretary, 5225 Spruce St., Philadelphia, Pa.

Pennsylvania State Veterinary Medical Association. Lancaster, Pa. October 26-27, 1933. Dr. Thos. D. James, Secretary, 816 N. Main Ave., Scranton, Pa.

Florida State Veterinary Medical Association. St. Petersburg, Fla. October 30-31, 1933. Dr. J. V. Knapp, Secretary, The Capitol, Tallahassee, Fla.

Twelfth International Veterinary Congress. New York, N. Y. August 13-18, 1934. Dr. H. Preston Hoskins, General Secretary, 1230 W. Washington Blvd., Chicago, Ill.

ADDRESS OF THE PRESIDENT*

By N. F. WILLIAMS, *President*
American Veterinary Medical Association
Fort Worth, Tex.

In complying with that section of our constitution which requires your President to deliver an address upon this occasion, I have chosen to review hastily the history of our profession and that of the live stock industry, that we may better estimate our hope of survival.

There can be no virtue in attempting to conceal the fact that the veterinary profession is suffering sympathetically the pains of agriculture that languishes in the depths of despair, a victim of the unwise counsel and misdirection of political forces that are no longer manageable. Unfortunately, the public generally rises in popular acclaim and approval of the destructive forces that fatten on the wreckage of the things that they destroy. It is the spectacular things of life that appeal to the proletariat. Being of constructive purpose and practices, veterinary medicine is not spectacular. It has unpretentiously, from the beginning of time, stabilized agriculture by the service it has rendered to the live stock industry. It is questionable if any profession, art, or craft is of greater antiquity or has rendered more consistent and valuable service to humanity. No thoughtful person can subscribe to the proposition that our profession is of lowly origin, except as the origin of the entire human race may be so considered. Where did that profession come from? What of its origin?

A somewhat poetic answer might be—"Out of a long, long ago, as a part of evolution or advancement, it has paced with the things that revolve about it and need it most. From out of the mists of mysticism, thread from thread has been untangled, until in orderly, scientific exactitude, it functions without delusion." In support of a claim to antiquity, it is not necessary to theorize upon the possible happenings of prehistoric times.

Ancient records of events, some of which are inspired, are freely accepted as competent evidence in cases coming within their purview. The history of mankind is said to be the story of his insane desire to destroy himself, and of nature's efforts to save him. It is the story of individual struggles, of the rise and fall of governments, the frustration of personal ambitions

*Presented at the seventieth annual meeting of the American Veterinary Medical Association, Chicago, Ill., August 14-18, 1933.

and the instability of positions of distinction. Regardless of all that, Adam still retains the distinction of being the first human being upon this earth. He was an agriculturist with a balanced program. His son Abel, the actively functioning live stock force of that day, was sufficiently proficient in the selection of superior animals from his flocks as to merit the approbation of the Divine Creator. His practice of regularly drifting the herds and flocks to clean pastures and pure waters, an approved method of minimizing infestations and infection hazards, suggests a knowledge of veterinary sanitation. It is not unreasonable to assume that one so intimately concerned with animal life and so absolutely dependent upon his own resources, rendered aid to his charges in time of distress, especially at the lambing period, or when minor ills and injuries were presented. If this be acceptable, the rudiments of veterinary practice were surely incorporated in Abel's live stock program.

Concentration of Animals Favors Disease

Concentration of human or animal population in a limited space favors the incidence of disease. As the concentration is intensified, the condition becomes increasingly acute, when, in the absence of sanitary compensation, disaster inevitably follows.

The biblical account of Noah and the Ark records the most intensive animal concentration that has ever occurred on earth. That Ark, with its living cargo, rode the waters for one hundred and fifty days, finally disembarking the same number and the same identical living things that had been taken aboard. There was no death loss, no impaired potency or other detrimental sequelae; but there was an unmistakable implication of applied live stock sanitation in the presence of the most intensive live stock concentration of which there is any record. As the various living things involved in that historic event were the only source of the life with which we are now concerned, a sense of familiarity and nearness seems to minimize the distance to that time of long ago. In face of the record, we cannot escape the conviction that, somewhere in that cargo, the accumulation of veterinary experiences since the time of Abel were preserved. Taking root, they started anew, the march of veterinary medicine in step with the advance of agriculture.

Evidence of further and indisputable progress is found in the Code of Laws that Hammurabi, the wise King of Babylon, gave to his people two thousand years before the Christian Era. Those laws, the oldest in the world, and which made the Babylonian

State the best administered empire of ancient times, contained a section which prescribed the fees that the doctor of oxen and asses should receive for service and provided a penalty for malpractice. Malpractice, be it remembered, is aptly defined "as the conduct of any profession in an illegal or wrong way," the word "profession" being essential to the definition. Here indeed is sound corroborating evidence that our profession was recognized four thousand years ago.

Basis of Modern Meat Inspection

Some five hundred years later, we reach the time of Moses, the first great teacher and the civilized world's first author, he who wrote the story of the Creation. Among the accomplishments of the versatile, indomitable Moses, a knowledge of physiology, hygiene, sanitation and parasitology, which he utilized most effectively, was not the least. He did expedient things and made no note of reward, or of punishment hereafter. His laws were designed for the immediate guidance of his people and with due regard for the materials with which he had to deal. That section of the Mosaic Law, wherein he tells his people what to eat, was the basis of our modern federal meat-inspection system, and furnishes the groundwork for all present food-inspection service.

Little changed from the time of the patriarch, orthodox rabbis even now observe the slaughter and inspection practices of old, as a protection to a distinct and peculiar people who constitute the nation founded by Moses long ago; and who still look to his laws for special daily guidance. Moses was familiar with all the phases of life on the range, as he undoubtedly was with the Hammurabi code.

His experiences, when forty years of age, with the flocks of Jethro which he herded to the far side of the desert, no doubt developed that fine discernment which enabled him to protect his people from diseases and parasitisms of animal origin.

That wise old leader who founded in ancient times a meat-inspection system that still endures, he who successfully guided the children of Israel through forty years of wandering, avoiding or compromising every hazard of mixed animal and human populations, certainly applied the science of veterinary sanitation in a most practical manner. It is highly probable that veterinary practitioners were numbered among the unmentioned advisors of Moses during his memorable pilgrimages.

Ancient Greece placed the veterinary art with the general pursuits of medical and surgical knowledge, and the Romans

held it in high esteem, as is shown by her medical and agricultural recorders. During their domination of Spain, the Moors bestowed solicitous attention upon it.

It is significant that, in the ancient time, when the welfare of the domestic animals was the most important consideration of the various communities, veterinary medicine ranked among the highest of all professions.

The downfall of Rome plunged the highly cultivated art, together with all other branches of learning, into the oblivion of the dark ages, from which it would seem to have emerged by way of the farrier's forge, when in the Middle Ages the iron shoe was adopted as a protection for the horse's foot.

All of the present diseases of live stock existed in the valley of the Nile in ancient times, as is attested by the record of pestilences, plagues and scourges that raged there. Tuberculosis, infectious abortion, anthrax and rabies, with all of which we still are concerned, flowed from the fountain-head of civilization eventually to engulf the world.

Rebirth of Veterinary Medicine

Veterinary medicine had its modern rebirth in 1761 when, aided by the French king, Francis I, and the government, the veterinary college at Lyons was founded, to be followed in 1776 by the institution at Alfort, near Paris. Subsequently, additional schools were founded in France; other European countries followed the example until almost every nation had a government-supported veterinary college in operation. After several unsuccessful efforts, the present Royal Veterinary College was founded in London, in 1791, and was warmly supported by King George III. Although the kingly urge may have been for the salvaging of horses wounded in wars, a more logical incentive may be discovered in the live stock improvement operations of that period, when breeder competed with breeder and nations strove for supremacy. In England, the modern improvement of cattle did not begin until 1745, after the systematic culture of higher quality grasses was inaugurated.

This was the age of Robert Bakewell, who has been distinguished as the patriarch of all the generations of animal breeders since his time. Disregarding all preconceived notions governing the production of farm animals, he was the first animal breeder of record to show that the concentration of blood elements is a ready and most effective method of establishing and fixing desirable characteristics. His great success was with long-wool Leicester sheep and Longhorn cattle.

As evidence of the early value of this improved blood, it is stated that one of his celebrated rams earned at stud, in a single season, the sum of 800 pounds, equivalent to $3,888, based upon the pound sterling in prosperous times. At the Oxfordshire sale in 1791 (the year that the Royal College was founded), several of the Bakewell Longhorn bulls sold at auction for more than 200 guineas each, or upwards of $1,000 a head.

Other breeders adopted Bakewell's methods, notably Charles Collin, who later sold his herd of 47 improved Shorthorns at auction for $35,000, the top bull going at the unheard of price of $5,000 and the choice female at $2,150; the balance of the herd averaging approximately $619 a round.

New Era for Live Stock

In Herefordshire, other breeders were equally successful in establishing the well-known Hereford breed of cattle, as they also were in the sale of surplus stock at consistently lucrative prices. The high quality sheep which were in great demand for flock improvement purposes found eagerly bidding markets of unprecedentedly high prices. A new era had dawned for live stock. The almost fabulous sales of improved animals of fixed type, and capable of transmitting that type, gave leaven to the modern stock-breeding industry that has achieved undreamed of success.

An independent point of interest is disclosed in this sketchy account of the moment, when modern veterinary medicine was cradled in the now age-mellowed institution at London, England. It signifies that Robert Bakewell, a practical breeder, positively and profitably demonstrated the operation of the law of heredity at least half a century before Gregor Mendel, the noted biologist, who is popularly credited with that discovery, was born.

Further, that practically all of the improved European breeds had been established as a result of that success and in accordance with its methods. In fact, specimens from the improved herds and flocks had already been exported to various parts of the civilized world, including America, many years before Mendel discovered the Bakewell breeding principle through the medium of peas.

Meat always has been an indispensable item in the human diet and meat-eating nations have been the dominant powers since time began. As the human family spread in ever-widening currents eventually to cover the earth, live stock was its sustaining

power. Coincidentally, animal diseases (older by far than the birth of civilization, likewise were scattered in all directions.

The first live stock on this continent were the few Andalusian cattle, and the seventeen horses landed by Cortez near Vera Cruz, Mexico, in 1515. As a result of subsequent importations and the natural increase that luxuriant ranges and ideal climatic conditions of the semi-tropical region of the Southwest favored, cattle herds of twenty thousand head, in the ownership of one person, were not an uncommon thing when, thirty years later, writers on agricultural subjects visited the great breeding grounds.

As the restless settlers pushed onward in the wake of the Franciscan missionaries, the lines of extension took a northerly direction along the Pacific Coast; another north to Arizona, New Mexico and Colorado; and still another, and the most important, followed the Gulf Coast to the coastal plains of Texas, founding the wild Texas Longhorn tribe.

From here the live stock industry of the Southwest spread as the ranges vacated by the buffalo became available; and the stocking of the middle west and northwest ranges, started in the early sixties, to reach its peak in the late eighties, was in progress.

When the herds going north from Texas met the eastern semi-improved cattle which had overflowed the Alleghenies and invaded the Great American Desert, losses among the latter from tick fever, to which the southern cattle were immune, presented the second serious live stock disease problem that this nation was called upon to combat.

Importations of Cattle Begin

Importations of cattle on the eastern coast of North America began with a few cattle and hogs that were landed by Portuguese fishermen and explorers on Cape Breton, in 1545, thirty years after the landing of Cortez in Mexico. These later were moved to Newfoundland and to the mainland. French, Dutch and English colonists, arriving at intervals, also brought their native stock with them. In 1611, horses, sheep and goats were imported into Virginia to replace the stock that was slaughtered to save the colonists from starvation the previous winter. In 1625, Dutch settlers of New Amsterdam brought with them 103 head of cattle. Four years later, six ships loaded with settlers and live stock, including horses, were landed at the Bay State

Colony. Finns planted a colony along the Delaware, stocked with sheep, cattle, hogs and horses.

The live stock of the East suffered from the severe winters which they were forced to endure without shelter, other than that of the woods, and were sustained by what they could find in the way of forage.

As areas were cleared for cultivation, the live stock, especially the cattle, were forced to move on with the pioneers who extended the frontiers to the west. Crossing the Alleghenies, these cattle herds of Atlantic Coast origin merged with the Longhorns of the Southwest to establish our native range cattle. The northern cattle presented the problems of contagious pleuro-pneumonia, tuberculosis and infectious abortion; and the mixed herds a combined menace at a time when foreign markets were opening to our live stock and meat products.

Superiority of American Hereford

The improvement of live stock in America, by the use of pure-bred importations, began sometime after the Revolutionary War and was scarcely under way until 1833, placing the rise of the modern animal breeding industry within the accomplishments of A Century of Progress. Stock from the Old World, especially England, has been freely drafted and so well utilized that today the American Hereford is superior to the specimens of the breed remaining across the water. This is not true, however, of the other breeds.

History repeats itself. The same conditions that led to the establishment of veterinary schools in Europe brought forth similar institutions in America, early among which was the Massachusetts school, in 1858, and the New York Veterinary College, in 1859. The first permanent veterinary schools were founded in New York, in 1865, and at Toronto, Canada, one year later.

The United States Veterinary Medical Association was organized in 1863 by veterinary practitioners, the majority of whom were self-educated men, there being no American graduates at that time and but few foreign graduates in this country. With the motto—"Non nobis solum (Not for us alone)"—as a guide, that modestly founded association has become the largest and most powerful veterinary organization in the world. During seventy years of useful activity, it has contributed materially to the substantial accomplishments of A Century of Progress that is being celebrated in this city today.

In answer to the challenge of diseases that threatened to annihilate the growing live stock industry, the Bureau of Animal Industry, a strictly veterinary organization, was established in 1884 and has conquered or has well in hand the old menacing plagues of antiquity. This great, smoothly functioning Bureau has kept the channels of trade open for American commerce, and has protected the breeding industry from foreign diseases by the careful inspection of arriving cargoes, and by sound rules and regulations that prohibit importation of live stock from dangerous areas. Among the noteworthy accomplishments of the B. A. I. are the eradication of contagious pleuro-pneumonia of cattle; the stamping out of dreaded foot-and-mouth disease on many occasions; and the obliteration of fowl-pest. The eradication of tuberculosis among live stock is already more than half finished and the areas reclaimed from the fever tick exceed the combined area of several European nations.

Practitioner a Sound Foundation

Every branch of veterinary science has been highly developed in America since 1863; and many discoveries, beneficial alike to agriculture, industry and the betterment of public health, have resulted. That veterinary superstructure erected through the years attests the soundness of the practitioner-foundation that supported it. But, just as the veterinary art, upon which the ancient world bestowed distinction, toppled from the heights of prominence and was lost to the world for ages, so will modern veterinary medicine fall unless the security of its foundation be zealously preserved.

As every organized human activity carries within its bosom the seeds of its own destruction, we must be watchful that no branch of our profession is permitted so to forget its obligation to the organized whole, as to become predatory in promoting its own interests; for that would weaken the tie that binds it securely to the foundation that the practitioner builds.

One of the greatest aids to the practitioner, and a weapon with which he can eventually emancipate himself, became available when the "veterinarian-controlled veterinary supply house," dedicated to the policy of "sales to veterinarians only," was developed from a dream to living reality by that stormy old champion of the rights of the practitioner, Dr. Hans Jensen.

In the present time of stress, the practitioner's most faithful and uncompromising supporters have been the ethical veterinary supply houses and the veterinary journals, which long ago

pointed out the danger of temporizing with those whose methods and practices tend to undermine the profession.

The editorial policy of the JOURNAL of the A. V. M. A. has reflected the opinion of your President and your Executive Board, as ably written by your incomparable secretary, Dr. H. Preston Hoskins.

Your Executive Board is a well-balanced force of indefatigable workers, many of them seasoned by years of service in your behalf. Members of the Association should remember that in every organization of special units, those interests which are actively urged by interested members usually fare better than do those left for one person to foster. Unless the demand for reforms, changes, modification or action of a given character is sufficiently urgent to evidence popular demand, they will not receive the consideration that they might otherwise merit.

COMMITTEE WORK IMPORTANT

To the various standing committees of your organization is delegated the task of appraising the activities of its various specialized professional units, and of recommending changes of policy, practice or standards in the interest of the unit concerned and mindful of that unit's filial obligations to the profession at large.

Special committees are created to cope with special problems, usually of more or less perplexing character. On occasions they are our diplomatic representatives who meet with and discuss points of contention with the representatives of conflicting forces or organizations, in an effort to promote harmony. All too often, the only reward that comes to him who serves is the consciousness of a duty well performed. Impressed with the fact that there is greater security in the composite judgment of a thoughtfully selected committee than there could possibly be in the judgment of an individual, your President has neither interfered with the activities, nor attempted to anticipate the reports, of any of the committees.

As committee reports are all-important, and self-interest a poor interpreter of the wider organization needs, it is essential that such reports should be acted upon by a numerically encouraging and representative membership of this organization. The business policy of this Association is the concern of its every member. The soundness of that policy will at all times depend upon the serious interest that each individual member displays

in formulating such policies. To the committees that have labored so faithfully, appreciation is here expressed.

The Special Committee on Agricultural Extension Service is given special consideration here, because it is dealing with a situation wherein agriculture has been prostituted, business in general undermined, and the life of the veterinary practitioner threatened by the unlawful activities of a tax-supported organization that no longer observes the law that created it.

Land-grant colleges are contemporary with the birth of the A. V. M. A. In answer to the crying needs of agriculture for guidance and orderly expression, those two great institutions took form, almost simultaneously, and for many years labored in perfect understanding, each in its separate sphere. Each in its own way met and overcame the obstacles which, from time to time, beset it. Frank challenges and open attacks have the virtue of warning. The hard blows fairly struck are soon forgotten; it is the silently working, insidious undermining of the craven-hearted schemer that all fair-minded men condemn. When sharp practices creep into the halls of learning, when the unclean hands of mercenary politics touch its portals, the fountain of knowledge is poisoned by the virus of lust.

Rise and Fall of Agriculture

During the past twenty years, we saw an agriculture that was progressively unfolding, suddenly soar to the loftiest peaks of prosperity; we saw it suspended there in all the glory of golden opulence, even after the value-supporting structures had been swept away by the stroke of the pen that is mightier than the sword. We saw it topple from its giddy heights and today the echo of that crash reverberates in every hamlet of our nation. Statesmen shuddering at the threatening glance from beneath the knitted brow of professional politicians, and further intimidated by organized predatory interests, aimlessly probe in listless effort to discover the cause of it all, and the reasons for its protraction.

Roll back those twenty years and start your investigation in the land-grant colleges to see how religiously the purposes of the Act which gave them birth have been honored in the meantime. Examine the various records in a truly businesslike way to determine just how faithfully the various funds have been devoted to the sole purposes for which they were appropriated. Check the existence of each department against the acts that govern the institution and dignify it with the stamp of legality.

Look well to the days when those institutions, under the pretense of aiding the farmer, entered the field of biologic production, promoting, through members of its staff and the county agents, the sale of unsupervised, uninspected products, in competition with legally established, reputable commercial concerns whose production operations are rigidly supervised by representatives of the federal government, and whose products, positively identified, were released for distribution only after careful tests and examination. Forget, if you will for a moment, the unfair competition phase, and check any remaining records of those early intra-institutional industrial plants, in an effort to determine whether all of such plants were invariably operated in the interest of the farmer, to what extent the farmer profited and whether the income from the activity was in whole or in part diverted to other purposes than biologic production in the interest of the farmer.

Commercialism in State Institutions

Regardless of my personal conviction that no state institution has a semblance of right to engage in the production or sale of any commodity in competition with organized business, I do not desire my remarks on this occasion to be misinterpreted by the members of this Association or the general public, as a blanket indictment of all the individuals who operated such plants as employes of offending institutions. I know, of my own knowledge, that many veterinarians, who were so situated, were too far above duplicity to indulge in or knowingly tolerate any irregularity of which they were cognizant. I do, however, include in the indictment every political trickster who would despoil an educational institution by subjecting it to the indignity of irregular and sordid commercialism. Once an individual or an organization has deliberately transgressed the law, the rules of business, or the observances of decency, the temptation to repeat the digression becomes more difficult to resist. In my judgment the activities of the county agent in the field of veterinary practice may be traced rather conclusively to the sale and distribution of land-grant college-produced anti-hog cholera serum. Encouraged by conniving superiors, he was so emboldened as to widen his lines of merchandising and spice his irregular activities with a pretense to veterinary knowledge which he foisted upon the trusting farmer, who believed him to be (at least in the early days) the reputable representative of the federal government discharging a mission of philanthropic nature.

To the exploiter, the year of 1914 was fraught with possibilities. The Extension Service dug itself in by capitalizing the fancied necessity of super-increased production of agricultural commodities in the hysterical hour of a foreign war in which we were not immediately concerned. True, the warring nations did present an immediate and insatiable market for materials that could be furnished at our own price. The imperative needs of that occasion raised commodity values sufficiently to insure increased production without the aid of the county agent. Long after the soldiers had returned to their homes, even after the grass had covered the scarred battle fields, the Extension Service program of "increased production" was diligently prosecuted, surplus was added to surplus, values sank to ruinous levels, assets became frozen and the business of the nation fluttered at lowest ebb. The diagnosis was "over-production" and the prescription, "destruction of surplus crops in the fields," destruction of that extra blade that had been made to grow where only one had grown before.

Increased Production and Its Antidote

The Extension Service, which has always taken complete and individual credit for increased crop production, is now busy administering the antidote. Were it compulsory for the farmer to destroy any part of his stored or growing crops without adequate and prompt compensation, the agricultural extension service would most certainly have evaded execution of the order. As arranged, the county agent now comes to the farmer in the guise of a faithful benefactor with funds to pay him to do what, to every thinking farmer, must appear most inconsistent—destroy the surplus that the same county agent had but yesterday persistently urged him to produce.

A careful and critical review of the last twenty years will disclose that county agents have ruthlessly invaded many fields of business, that they have initiated and sponsored the organization of special groups for political and mercenary purposes, to the detriment of agriculture, legitimate business and the veterinary practitioner. It will further disclose the deplorable fact that in some instances they have received comfort from individuals within the ranks of the veterinary profession. It will disclose a most unusual situation, wherein, despite the depression, the entire Extension Service program suffered little if any material financial embarrassment. Salaries, always extraordinarily high in comparison with those of the regularly and long established federal bureaus which are governed by Civil Service

regulations, remain unimpaired. These have been twenty busy years for the Extension Service. In that period they have practically taken possession of several land-grant colleges and would appear to be devoting them to enhancing the prestige of the Extension Service and of strengthening its political ties.

If it is true that men fall victims to their own facility, then extension worker after extension worker, intoxicated by his own seductive practices, has been lured to further conquest. With "Four-H Boys Clubs," "Increased crop production," and "Surplus crop destruction," as alternate and convenient rallying cries, he prowls in the field of business, creating discord and hastening the day when the people of this tax-impoverished nation will rise in desperation and demand a return to the form of government which those who wrote the Constitution intended, and which tolerates no such abuses. And, what is our remedy? The Committee on Agricultural Extension Service has accumulated sufficient evidence and presented a brief, so convincing, that it is inconceivable that the Congress which will convene in January can longer permit this heedless, unmanageable group to carry on.

Having joined other aggrieved business groups in general protest, the cause of the veterinary practitioner is no longer a matter of individual concern, to be dismissed as unimportant by any body of competent jurisdiction even if so inclined. Further, this Association is by its Constitution bound to protect and promote the professional interests of the veterinarian.

Professional Interests Must Be Protected

The practitioner is the element aggrieved and the practitioner, be it remembered, is the foundation that supports the superstructure which represents those engaged in all other veterinary activities.

Veterinary medicine is founded on the practical experiences of the men who, from the very beginning of things, on through generations of scientific unfoldment, have been directly in contact with field problems. He who clinically observed the subject, by some means reached a diagnosis and applied a remedy, carried the torch and was our sustaining force in every age. When the ancient science of veterinary medicine was submerged in the dark ages, its superstructure was the first to go. Its vital spark found refuge in those of the field of practice, as the life of a plant finds sanctuary and preservation in its adversity-resisting seed and roots.

Truly no one will deny that the practitioner is the foundation upon which the veterinary profession rests. Any circumstance

or combination of circumstances that interferes with the practitioner's independence of operation within that field must weaken the entire structure. He has always been the contact man for our profession; he must deal not only with the diseases and parasitisms of live stock in a practical and scientific manner, but with the general public as well. Be it remembered that the general public is a mass group of varied interests, impulses and prejudices and still bound to the lingering superstitions of an age that enshrouded medicine in mystery and magic as it marched down the musty centuries, even to modern times.

The veterinary profession offers neither ease nor affluence; rather it exacts that he who qualifies shall consecrate himself to the utilitarian and humanitarian tasks of promoting the welfare of animal and human life, by rendering sympathetic, competent service to those who depend upon him.

Practitioner Has a Responsibility

The activities of the practitioner preclude set rules of conduct. He must be guided by the high ideals of those who have given so freely of their substance, time and effort, and whose exemplification of rectitude and fairness has sustained the profession in every crisis. To be grounded properly, the practitioner should be familiar with the history of his profession and with the origin and development of the live stock industry that it serves. A study of these will strengthen his confidence in himself. It will convince him that his is a special and indispensable service, without which the entire scheme of civilization would crash. It will enable him better to understand the character of service needed, and will guide him in equipping himself to render that service better than can any other person.

As veterinary medicine is not philanthropic, business methods must govern its operation. The practitioner's right to a profit must be based upon a scientific service rendered in a truly business and professional way. Sound business is based upon sound practices. Costs, inclusive of all overhead involved, are regularly determined for the purpose of establishing the basis for the reasonable profit that a given business venture merits. Material differences in the selling price of various commodities of like character are generally due to inferior quality of merchandise or to the perverseness of irresponsible manufacturers or dealers who, operating upon the verge of insolvency, constitute a menace to all sound business.

Price cutting is not merchandising, nor professional fee cutting to the credit or ultimate profit of him who indulges in it.

It is desirable in this hour of business restabilization that the practitioner should follow the suggestion of our national government and, through the medium of his local society or association, reach an understanding as to reasonable fees for various phases of practice, to the end that the prestige of the profession may be rehabilitated in all localities where loose professional practices and unfair and illegal competition have impaired it.

Every profession has its exploiters, its cowards and its traitors. Professions are finding means to purge themselves, as a matter of fact, of such undesirables. In fact it is the solemn duty of every profession as a matter of self-preservation and in justice to the public to which it caters, to clean its ranks in no uncertain manner.

In addition, the practitioner must cultivate his field, mindful that every veterinary service that is slighted or neglected by those of the profession, will be avidly seized upon and exploited by an increasing band of pirates and fakers who, in the guise of protectors, prey upon agriculture even in her hour of deepest depression.

Veterinarians Must Put Their House in Order

In final analysis, the veterinarian must order his own house, meet in a business and professional manner every demand for legitimate service that comes within his field of usefulness. He should meet his civic responsibilities graciously, not as an agitator or malcontent, and support the activities that go for good government and a contented people. Every veterinary practitioner should be a member of this Association, which exerts its efforts in his behalf.

True, the clouds of adversity have frowned threateningly upon the field of practice, predatory interests have encroached upon it, and the groans of stricken agriculture come to him night and day.

The plight of the practitioner is even now reflected by disturbances in other branches of the profession. The superstructure is trembling as does the leaning tower that falls. We must devote our every energy to repairing the foundation, to clearing from the field of practice all of the accumulated unfair competition and annoyances that deprive the practitioner of his rights to a livelihood. We must recognize the enemy of the practitioner as the common enemy of all, and join in a concerted effort to destroy him.

There must be a general stirring up of branches, and as all reforms come out of revolution, it is here suggested that, as an

inducement for all practitioners to join this Association, your President hereafter be elected by popular mail ballot, as are your Executive Board members. This would afford practitioners, who cannot regularly attend meetings of the Association, an opportunity to cast their votes for the men of their choice for this high office.

The JOURNAL should close its advertising columns to those firms considered unethical because of offending sales policies.

The experiment station worker should not extend his activities beyond the legitimate limits of his enterprise.

The regulatory departments must remember that theirs is to control and eradicate infectious and contagious diseases and to protect the live stock of their respective communities from menaces from within and without the state of their jurisdiction, and this does not in any manner imply research endeavor nor a right to interfere with the practitioner's legitimate operations.

The veterinary college staff is maintained for a specific purpose and does not comprehend any activity that would interfere with the man in the field of practice, nor with the research worker, nor the regulatory forces.

All of these activities are supported by the tax-paying public, of which the veterinary practitioner is a part, and do not comprehend any duplication of effort.

BRANCHES OF VETERINARY MEDICINE INTERDEPENDENT

Each specialized branch of veterinary medicine is presumed to be needful to the others. The possible prompt coördination of any set, or all of these units, is the only assurance that the industry and commerce of this nation have that they will not be overwhelmed by an uncontrollable outbreak of some rapidly ravishing plague or pestilence such as has wrought havoc even in modern times in European countries.

As a comparison of values in the scheme of civilization, be it remembered that it became necessary to close the banks of this country not long ago, for a period, in the interest of a saner and sounder financial system. If all veterinary activities on this continent were to cease for that same period of time, this nation would not soon, if ever, fully recover from the blow. The heart of the profession is still sound. A great deal of thinking is going on. The practitioner is finding the courage to stand firm for his own rights and to fight his battles to a finish.

The golden rule of conduct alone can lead us out of our troubles. The sooner we realize that we are our brother's keeper, the sooner the dawn of contentment will shine upon us again, and the motto

that inspired the founders of this Association—"Non nobis solum (Not for us alone)"—and gave hope to the live stock industry, will not fall to pieces in our hands.

CONVENTION NOTES

From *Tennessee* there were nine in attendance: Drs. William M. Bell, Nashville; L. G. Brown, Franklin; D. Coughlin and M. Jacob, Knoxville; C. F. Delap, Springfield; J. H. Gillmann, Memphis; J. F. Kagey, Kingsport; J. E. Peters, Pulaski; A. C. Topmiller, Murfreesboro.

Arkansas, Florida and Utah each boasted three representatives present. From *Arkansas*: Drs. J. H. Bux, Little Rock; Walter Martin, Jonesboro; R. W. Williams, Eldorado. From *Florida*: Drs. J. G. Catlett, Miami; G. W. Lewallen, Saint Petersburg; W. M. Lynn, Orlando. From *Utah*: Drs. Hugh Hurst, Salt Lake City; Osborne G. Larsen, Logan; N. C. Spalding, Provo.

Three states were credited with a pair of members in attendance: Drs. J. J. Flaherty, New Haven, and I. R. Vail, Bristol, upheld the honor of *Connecticut*. Drs. E. A. Birmingham, Dover, and C. C. Palmer, Newark, did the same for *Delaware*. Drs. A. L. Edmunds, Franklin, and Fay F. Russell, Concord, looked after the interests of *New Hampshire*.

Veterinarians to the number of eighteen registered from *Nebraska*. The list included: Drs. G. H. Anderson, Holdrege; Frank Breed, J. B. Lavender, E. H. Meyer, Carl J. Norden, W. T. Spencer and J. E. Weinman, Lincoln; A. C. Drach, Paul L. Matthews and D. C. Murdock, Omaha; P. L. Cady, Arlington; M. L. Cline, Plainview; B. F. Lott, Gresham; H. C. Petersen, Marquette; C. J. Sall, Bertrand; P. Simonson, Hooper; W. A. Walther, Moorefield; F. E. Ziegenbein, Nebraska City.

Texas veterinarians showed their appreciation of having the Presidency with an attendance of twenty-two from the Lone Star State: Drs. S. G. Bittick, R. C. Griffith, B. L. Jacobs, Frank R. Jones, Frank D. Porter, H. V. Cardona, John Ramsey and N. F. Williams, Fort Worth; P. W. Burns, R. C. Dunn and Hubert Schmidt, College Station; Major C. M. Cowherd and Major Lloyd C. Ewen, Fort Sam Houston; Drs. M. E. Gleason, San Antonio; O. M. Franklin and E. F. Lanham, Amarillo; I. B. Boughton, Sonora; Col. Robert J. Foster, Fort Bliss; Drs. Frank Hecker, Houston; T. O. Scott, Waco; Richard A. Self, Dallas; J. T. Traylor, Harlingen.

ECONOMIC ASPECTS OF VETERINARY MEDICINE*

By JOHN R. MOHLER, *Washington, D. C.*
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With this and other nations grappling with huge economic problems affecting millions of people, the interests of any group of workers must, of necessity, be a minor part of the entire program. Our veterinary profession, numbering scarcely 12,000 active workers, exerts, it is true, great influence on our live stock resources and public welfare. But though recognized by us and related groups, the value of veterinary work may seem remote to persons who are unemployed, discouraged, and hungry.

The present public mood is centered in economics and is looking toward relief from that source. The national atmosphere is surcharged with the relationships between labor and capital and attendant questions dealing with the maintenance of life itself.

At first glance some of the changes that have taken place in recent years appear to be paradoxical. Less than two decades ago, the appeal was to produce more and eat less wheat, meats, and some other commodities. Today the situation is the reverse —to produce less and consume more.

In our profession, events that would surprise a veterinary Rip Van Winkle likewise have transpired. The practitioner who now derives his livelihood from equine practice is difficult to find. Yet many in this audience can remember the time when this item constituted 90 per cent of their business. The prevention of disease through the use of serums, vaccines and bacterins has been increasing. The pig has gone sanitary, and the lowly chicken is becoming a frequent candidate for skilled veterinary service. These seemingly erratic changes, nevertheless, are in accord with well-established economic laws which may be studied and utilized just as we do those of chemistry, biology, and other sciences. It is the part of wisdom for veterinarians to adapt themselves and adjust their affairs to the constantly changing order of things. In the federal Bureau of Animal Industry we have had some experience along this line, which may be helpful to others.

ECONOMIC LAWS INFLUENCE QUARANTINE POLICIES

The Bureau's policies have an economic as well as a scientific foundation. The well-known procedure of stamping out foot-

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and-mouth disease by slaughter is based on the fact that it costs less to destroy the virus by this method than to live with it. Official data show that in the United States the cost of eradication by the slaughter method—including all indemnities and operating expense—is a very small fraction of what the annual loss from the same disease would be if it were enzoötic here.

For the same reason the risk of introducing foreign diseases by permitting unrestricted importation of live animals, fresh meat, hides, skins, and many other products from countries where dangerous infections exist, outweighs the value of such importations. Thus, live stock quarantine regulations have not only a scientific basis, but a further economic sub-base or general foundation.

Comparatively speaking, our animal industry has attained a high plane of health and excellence through years of diligent effort of nation-wide scope. It would be questionable economics as well as unsound veterinary procedure to relax any form of preparedness that has proved to be valuable. The long-time general welfare deserves greater consideration than a short-time stress. We must consider the full cycle of economic changes and established scientific principles, if we are to avoid dangers that are not apparent in portions of an economic cycle.

From the foregoing it is apparent that the policy adopted rests on both the well-known law of self-preservation and that of supply and demand, broadly considered. In the simpler problems, however, of local or regional scope, a temporary change in established regulations may sometimes be both desirable and sound.

Take a case in point. During the severe outbreak of hog cholera that occurred in the fall of 1926, the supply of serum became low. The Bureau regulations require now, as they did then, a period of about three weeks for testing the potency of the serum. But with the demand for it overtaking the supply, the abnormal situation required special methods of procedure. It was a case of putting on one side of the balance the risk of distributing untested serum, and on the other side the probable toll of hog cholera that would have resulted from slavish adherence to the regular procedure.

Emergencies Alter Procedures

Fortunately, the Bureau had reliable information which showed that the risk from untested serum would be much less than the probable loss of hogs from cholera if the protective serum was

not made and released promptly. Temporarily, therefore, the Bureau suspended part of the regulations and during the emergency permitted the distribution of untested serum, properly labeled, of course, to show that it was untested. Thus, the best biological practice was set aside, for a time, in deference to the more fundamental need of adjusting supply to demand. The action made available serum enough to treat 700,000 hogs about three weeks earlier than otherwise would have been possible.

In several lines of field work the Bureau has had to deal with the economic principle commonly called the law of increasing and diminishing returns. According to the operations of this familiar law, a certain expenditure of money, time, or effort gives a certain result, but doubling that expenditure seldom gives an exact double of the result.

For instance, if the amount of indemnity paid for tuberculous cattle were doubled, or if the field force were doubled, it is improbable that the volume of testing would respond in like proportions. In other lines of veterinary field work also, the results obtained are not necessarily in proportion to the financial allotment or size of force. There is a certain point at which best results are obtained in proportion to the investment in men and money. Determination of this point involves also the law of variable proportions in which seasonal influences, the character of the work, and, of course, the caliber of available men all play a part. To a large degree, these economic laws affect the distribution of Bureau personnel and expenditures for different projects. They operate, of course, in the direction of diminished as well as increased returns. Hence, too small a veterinary force for an essential duty may be as costly as an excessive number of men. The proper or ideal number is the goal to seek and maintain.

Insurance Features of Veterinary Science

Another fundamental that bears directly on veterinary work, is the distribution of financial risk. It has wide application in the insurance business and various other large enterprises. The underlying purpose is to minimize loss to the individual through a plan that gives protection at a price so low that a person willingly pays it. This concept underlies the use of dips and disinfectants. It finds application in the use of veterinary biological products that prevent disease. Various sanitary practices, involving, for instance, parasite control, are essentially forms of insurance. The outlay may be in labor, cash, or mate-

rials, but the principle is the same. The user elects to make a very small expenditure rather than risk a much larger loss.

A preventive veterinary practice that the Bureau now is requiring is the chlorination of effluents from tanneries which handle certain classes of imported hides. This safeguard has become routine procedure as insurance against the spread of anthrax from such sources.

A recent Bureau study of biologics produced under U. S. licenses and permits shows a general upward trend in the kinds used to prevent disease, or to diagnose it before outward symptoms are apparent. The quantity of anti-hog cholera serum produced and distributed is now about five times as great as in 1915 and 1916, shortly after commercial production began. In the last dozen years, the output of canine distemper serum has increased about tenfold, of rabies vaccine more than a hundredfold, and that of some poultry biologics even more. The production of tuberculin has increased greatly in the last 15 years. New biologics are constantly being developed and produced.

Regarding the use of disinfectants, adequate comparative data are lacking. But in the number of producers of officially permitted disinfectants there has been a distinct upward trend. The list of such producers ten years ago contained 27 names. This year the list reached a total of 117. The increase of more than fourfold indicates a development in the direction of better live stock sanitation through disinfectants.

Statistics Serve as Compass

Still another phase of our general topic is the value of reliable information, systematically kept. The Bureau keeps current records of meat inspection, tuberculosis eradication, hog cholera and virus-serum control, tick eradication, and other major projects. These records serve the same purpose for the live stock industry as a chart and compass for a ship. They are vital for steering a safe course and for many administrative functions. Besides, some of the data have commercial value. Meat inspection records reveal current trends in the nation's meat supply. Incidentally, the federally inspected slaughter of hogs in June of this year broke all previous records for June. The Bureau's annual quarantine order and map, indicating the areas infested with the cattle-fever tick, are regularly consulted by officers of the hide and leather industry and by prospective purchasers of stocker and feeder cattle. Data on tuberculosis eradication are the basis for recognizing promptly those states and counties

which qualify for the modified accredited status. As is well known, such recognition is conducive to sales of breeding stock, simplifies interstate business in cattle, and is becoming a factor in the sale of milk and butter and in the location of creameries and cheese factories. Likewise, progress in tick-eradication work reveals and makes available areas suitable for dairy and live stock development with attendant uses of supplies used in such enterprises.

Besides this information, there are data from other official sources, of veterinary and commercial interest. Department of Agriculture estimates on the national live stock inventory at the beginning of this year showed a 2 per cent increase in the total number of live stock over the previous year, but a 17 per cent decrease in value. Since three years ago, the total value of live stock has been cut in half through decline in prices. The number of horses and mules on farms in this country at the beginning of the year was the smallest in 40 years, though prices of these animals recently have turned upward with prospects for some come-back.

Figures such as these seem to warrant study and appropriate action by the veterinary profession. When the value of a *normal* animal is low, that of a *sick* animal is naturally still lower. Such a situation has a negative influence on the service that a veterinarian is called upon to render. Yet to cast anchor and wait for recurrence of former conditions invites stagnation. We must accept constant changes as a matter of course, also keeping in mind that they follow economic laws. Adequate training, efficiency, and ingenuity should help in making the necessary adjustments to new conditions.

In recent statements Secretary Wallace stressed this point. "I doubt," he said, "if scientists have considered, as much as they should, the impact of the present economic situation upon science." He remarked further, "Scientific experimenting long ago caught the fancy of the American people. Hundreds of thousands of farm boys are born inventors. Now that the needs of the times are changing, it is to be hoped that the capacity for straight, clear thinking in the field of social inventiveness will grow."

Social and Economic Inventiveness

I have already mentioned typical cases in which the Bureau of Animal Industry has adjusted its work to changing requirements. In reviewing veterinary literature of the last year, I have noted with satisfaction the able manner in which some

members of the profession in private practice have set their sails to conform with the direction of prevailing economic winds. Scientific papers of several veterinarians have dealt with poultry work, with special reference to hatchery and brooder sanitation. Another interested himself in boys' and girls' club work and tested the calves of members in preparation for a local fair. In a group of alumni notes from one of the leading colleges it is significant that several veterinarians have engaged in the manufacture of biologics in keeping with the trend toward preventive veterinary service.

Merely to illustrate how these tendencies may be pursued to advantage, let us consider other possibilities. Much of the so-called disinfection of buildings and premises after outbreaks of live stock disease lacks thoroughness. In official work, disinfection is conducted under veterinary supervision. This suggests opportunities for alert practitioners to establish disinfecting services, thereby placing such operations on a more effective professional basis. Disinfecting and fumigating services are already well established for various human needs in cities, and the proposal is merely an extension of the same principle to live stock work.

In the treatment or prevention of certain diseases, especially those caused by parasites, centralized services may save many miles of travel and increase one's income. The tendency to organize and centralize veterinary service is already apparent in the community treatment of horses for bots and other internal parasites. The underlying principle is to assemble animals at convenient points and treat more at a lower cost per animal, thereby benefiting both the stockman and the veterinarian. It is in line with the procedure in much official dipping and tuberculin testing, also with the poultry clinics held in avian tuberculosis eradication. This type of service seems at least to have some possibilities. Possibly our veterinary journals may see fit to devote occasional articles to matters of this kind and to economic trends that so vitally concern their readers.

These trends could be more readily seen and followed if the states collected and maintained more complete statistics regarding animal diseases, their prevalence and control, thereby supplementing the federal records. Some states already have recognized this need and, in spite of being handicapped by limited facilities, are obtaining very creditable results. But in many states, we must rely on opinions or estimates to a large extent. The state is a logical administrative unit and the development

of veterinary statistics, especially in a uniform manner among all states, should be highly valuable both professionally and commercially.

These remarks are offered not as a complete treatment of the subject but rather to suggest topics that may be developed more adequately by further consideration.

Summary of Salient Points

In conclusion the salient thoughts that I wish to leave with you are these:

Economic changes may be scarcely perceptible or they may be distinct—even violent. But in any case they are in accord with distinct economic laws. The operation of these laws affects the numbers and values of live stock, income of their owners, and the supply and demand for live stock products. Such changes thus have a profound effect on the veterinary profession and its services.

The trend of economic movements is studied readily and their principal effects may be anticipated with fair accuracy. Judging from observation and data on the manufacture of biologics and chemicals, veterinary medicine has been developed in recent years, especially along preventive lines.

This development suggests opportunities for mass treatment of animals or centralized services, thereby protecting live stock health at minimum cost to owners and extending the scope and usefulness of veterinary services. Disinfection of premises after outbreaks of serious live stock diseases preferably should be under veterinary supervision.

Changes in veterinary education and training should parallel the broader economic trends so that veterinarians may anticipate and adequately meet the developments, thereby keeping in step with progress. In addition to prevention and preparedness, the virtues of efficiency and ingenuity maintain their par values and pay dividends when given a reasonable chance to operate in any economic set-up.

The veterinarian has had an important place to fill in the economic life of this country. I am confident that he will always continue to fill it with credit to his profession and benefit to his fellowmen. Finally, let us include in our scientific philosophy the words of Bacon, "Nature is not to be governed except through obeying her." This quotation on the wall of A Century of Progress Hall of Science applies with special force to the economic aspects of veterinary science.

BLOOD STUDIES OF FOWLS WITH VARIOUS FORMS OF LYMPHOMATOSIS (FOWL PARALYSIS)*

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INTRODUCTION

Intensive studies by several investigators have revealed many interesting facts concerning lymphomatosis (fowl paralysis) but thus far the cause and mode of transmission have not been revealed.

Kaupp,[7] Doyle,[4] Pappenheimer *et al*,[9] and Johnson,[6] as well as others, have contributed to our knowledge, particularly concerning the pathology of this disease. The microscopic lesions strongly suggest that the disease is of an infectious nature, but as this has not been established definitely, it was thought that a study of the blood might reveal changes that could be detected in the early stages and that in this way it might be possible to pick out these individuals and remove them from the remainder of the flock. Leukemia and various forms of tumors in fowls have been studied extensively by a large number of investigators. These studies, however, have been confined chiefly to each specific malady and no relationship so far has been established between leukemia and the various forms of lymphomatosis. Since it has been found that small round-cell infiltrations of peripheral nerves, hyperplasia of visceral organs, iritis and lymphomatous tumors are lesions associated with the disease, it was thought that a careful study of the blood might enlighten us further on this increasing problem.

HISTORICAL

A thorough search of the literature impresses one that no work of this nature has been carried out in connection with this disease. Numerous investigators, however, have studied leukemia in its various forms. Schmeisser,[10] Warthin,[12] Stubbs,[11] and Moore,[8] report a marked increase of white blood cells in cases of leukemia and state that the blood appears pale, fails to clot readily, and is low in hemoglobin content.

In 1905, Butterfield[3] reported three cases of aleukemic lymphadenoid tumors of chickens, all of which showed marked enlarge-

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ment of the liver. The liver tissue was replaced almost entirely by masses of cells which resembled large lymphocytes of the blood of the hen. The type of cells, the periportal growth of the tumor masses, and the absence of increase of white cells in the circulation led to the diagnosis of aleukemic lymphadenoma or aleukemic leukoblastoma of the lymphocyte type.

METHOD OF PROCEDURE

This work involved the study of the blood of 101 fowls. Of this number, 45 were White Leghorns, 32 Barred Plymouth Rocks, 12 Rhode Island Reds, 2 Black Minorcas, 2 White Plymouth Rocks, 2 Buff Orpingtons, 2 Brown Leghorns, 1 Jersey Black Giant, and 3 were of mixed breeds.

The birds have been divided into four groups, *i. e.*, those with symptoms of paralysis of the limbs, but no gross lesions; those with symptoms of paralysis and gross lesions of lymphatic hyperplasia present; those with paralysis and tumors present, and those with gray eyes, associated with iritis and blindness.

The examinations have consisted in the determination of the coagulation time, which was accomplished by drawing 3 cc of blood from the wing vein into a 5-cc Record syringe. The blood was introduced immediately, in equal quantities, into each of three small tubes. The time was recorded from the time that the blood first entered the syringe until the blood in all three tubes was coagulated and the average time to coagulate was used for our data.

TABLE I—*Differential leucocyte count of normal fowls.*

AUTHOR	POLYMORPHONUCLEARS	LARGE MONONUCLEARS	LYMPHOCYTES	EOSINOPHILES	MAST CELLS
Ellermann and Bang	....	23	40	....	....
Goodall	37	....	....	6	1
Hedfeld	30	12	42	12	3
Mack	32.7	6.2	54.9	2.7	3.3
Schmeisser	29.6	19.4	42.3	4.3	2.2
Taylor	33	9	51	3.6	3
Warthin	21.5	14.5	35.5	10	2
Burnett	28.8	5.5	58	3.3	4.3
Average	30.3	12.8	46.2	6.0	2.7
S. D	± 4.4	± 6.0	± 7.6	± 3.4	± 0.95
rs	± 3.24	± 4.45	± 5.6	± 2.4	± 0.67
rm	30.3±1.01	12.8±1.68	46.2±2.09	6.0±0.94	2.7±0.26

The total erythrocyte counts were made by the use of standard technic using Toisson's fluid. The total leucocyte counts were made by use of the red cell pipette and physiologic saline, or Toisson's fluid in some cases, and by the method described by Blaine[1] in others.

For the differential leucocyte counts the blood-smears, for the most part, were stained according to Wright's method. Jenner's or Romanovsky's stain was used in a few cases. The hemoglobin was estimated in 13 birds by the Tallqvist scale and in the remaining birds by the aid of a Dare hemoglobinometer.

Results

Table I represents the average complete leucocyte counts of the six different investigators listed by Burnett.[2] These counts were used as the average for normal fowls in checking our work.

The following formulas were used to compute: (a) the standard deviation; (b) the probable error for a single observation and (c) the probable error for the mean.

$$\text{(a) S.D.} = \sqrt{\frac{\Sigma v^2}{n}}$$

$$\text{(b) } r_s = .6745\sqrt{\frac{\Sigma v^2}{n-1}}$$

$$\text{(c) } r_m = .6745\sqrt{\frac{\Sigma v^2}{n(n-1)}}$$

S.D. = Standard deviation
Σv^2 = Sum of the difference squared
n = Number of observations
.6745 = Constant

Since it is impossible to correlate these counts, with those made in this investigation, a comparison of the probable error of the mean is made in each case. It is evident, that as far as comparison is concerned, the probable errors of a single observation, as computed in this table, are of no real significance.

Tables II to XIX are self-explanatory, so only brief comments will be made concerning them.

An increase of large mononuclears is noted in table II as compared with the normal. The probable error of the mean is in nearly every case, as small as, if not smaller than, the normal. In no instance is there enough deviation from the normal to excite notice.

A slight increase in large mononuclears and mast cells is noticed also in table III. The standard deviations and probable errors agree quite closely with the preceding tables.

TABLE II—*Differential leucocyte counts of paralyzed birds with no gross lesions.*

BIRD	POLYMORPHONUCLEARS	LARGE MONONUCLEARS	LYMPHOCYTES	EOSINOPHILES	MAST CELLS
787	24.00	32.95	36.76	3.80	2.47
1517	25.30	21.83	43.84	4.50	4.50
718	50.00	15.19	26.14	5.39	3.26
1561	21.54	28.25	44.71	1.21	4.06
1659	30.66	16.03	42.68	2.00	8.01
1667	25.38	13.90	50.76	4.94	4.94
1718	53.94	14.06	26.97	3.85	1.15
1739	33.62	10.45	51.39	4.52	....
1702	37.33	16.00	36.08	4.08	6.48
1743	50.51	13.57	30.07	2.91	2.91
1738	29.20	12.52	52.81	2.71	2.71
900	31.71	18.01	41.98	2.34	5.94
1699	37.71	14.25	42.96	5.07	....
Dr. B.	44.37	12.45	26.16	10.23	6.82
1752	16.22	16.22	60.85	4.05	2.36
1753	13.51	14.52	66.38	2.18	3.37
45860	31.70	27.56	35.83	3.25	1.62
1888	34.00	12.00	51.00	1.00	3.00
1889	40.00	32.00	21.00	6.00	1.00
1890	46.00	36.00	16.00	1.00	1.00
1917	33.00	24.00	38.00	2.00	3.00
1922	30.00	28.00	40.00	1.00	1.00
1918	24.00	28.00	39.00	5.00	4.00
1919	41.00	19.00	36.00	3.00	1.00
1933	38.00	17.00	40.00	3.00	2.00
1934	22.00	23.00	49.00	3.00	3.00
1935	21.00	24.00	48.00	3.00	4.00
1937	32.00	21.00	42.00	2.00	3.00
1938	31.00	12.00	52.00	3.00	2.00
1939	47.00	14.00	30.00	2.00	7.00
1891	36.00	18.00	22.00	19.00	5.00
Average...	33.28	19.54	40.01	3.90	3.24
S. D.....	± 9.9	± 6.5	±11.4	±3.3	±1.9
rs.....	± 6.8	± 4.5	± 8.4	±2.3	±1.3
rm.....	33.28±1.2	19.54±0.7	40.01±1.4	3.90±0.4	3.24±0.24

The increase in large mononuclears and mast cells is likewise evident in table IV.

The large mononuclears and mast cells are high as compared with the normal. Table V shows a rather wide range in counts and, as the result of this, the standard deviation and probable errors are higher than normal.

Tables II, III, IV and V, of paralyzed and blind birds, show the leucocyte counts of each to comply rather closely. The large mononuclears and mast cells have been slightly high in each group.

TABLE III—*Differential leucocyte counts of paralyzed birds with gross lesions.*

BIRD	POLYMORPHONUCLEARS	LARGE MONONUCLEARS	LYMPHOCYTES	EOSINOPHILES	MAST CELLS
57576	14.33	12.67	68.42	1.43	3.11
57595	7.33	32.70	56.81	1.46	1.46
1627	49.64	11.31	31.93	5.11	2.01
1671	21.85	15.05	57.52	4.12	1.23
1723	45.20	13.58	30.06	9.02	2.12
1798	39.03	13.81	36.42	....	10.35
1799	41.69	15.67	41.69	....	0.93
H-I	41.75	18.99	33.33	2.32	3.58
H-II	27.31	18.20	39.43	3.94	11.07
45858	42.80	14.55	28.08	3.38	11.16
8	21.00	14.00	63.00	2.00	.....
1893	30.00	6.00	58.00	3.00	1.00
1906	31.00	27.00	40.00	1.00	1.00
1932	25.00	37.00	33.00	3.00	2.00
Average...	31.28	17.89	44.12	2.84	3.64
S. D.....	±12.03	± 8.26	±13.3	±2.4	±3.8
rs.....	± 8.43	± 5.73	± 9.24	±1.7	±2.7
rm.....	31.28±2.23	17.89±1.54	44.12±2.5	2.84±0.5	3.64±0.7

TABLE IV—*Differential leucocyte counts of paralyzed birds with tumors.*

BIRD	POLYMORPHONUCLEARS	LARGE MONONUCLEARS	LYMPHOCYTES	EOSINOPHILES	MAST CELLS
741	22.96	24.69	47.65	3.20	1.48
1649	51.30	19.66	22.03	3.08	3.90
1638	35.96	9.40	50.98	.....	3.69
1643	19.95	16.66	51.87	6.57	4.92
1669	8.09	27.32	43.11	20.24	1.21
1675	38.67	10.62	36.07	4.00	10.62
1782	28.72	15.02	53.49	.....	2.68
1781	42.23	23.37	30.21	2.17	2.17
1754	10.34	25.07	50.15	4.07	10.34
1866	68.00	8.00	19.00	2.50	1.50
1867	80.00	6.00	14.00	.....	1.00
1894	20.00	25.00	40.00	7.00	8.00
1907	29.00	34.00	31.00	4.00	2.00
1915	44.00	17.00	36.00	3.00	.....
1916	46.00	17.00	33.00	3.00	1.00
Average...	36.34	18.58	37.24	4.18	3.63
S. D.....	±19.1	± 7.7	±10.9	±4.8	±3.3
rs.....	±13.4	± 5.4	± 7.6	±3.4	±2.3
rm.....	36.34±3.4	18.58±1.3	37.24±2.0	4.8±0.9	3.63±0.6

TABLE V—*Differential leucocyte counts of birds with gray eyes.*

BIRD	POLYMORPHONUCLEARS	LARGE MONONUCLEARS	LYMPHOCYTES	EOSINOPHILES	MAST CELLS
1647	11.58	21.66	51.88	6.54	8.31
1692	43.96	23.12	30.74	.....	2.14
1708	22.55	21.24	53.75	.....	2.44
M-I	20.55	16.46	54.80	3.64	4.41
M-II	41.02	21.52	20.51	5.62	11.28
90	42.87	15.31	39.81	1.99	.....
1783	32.28	17.71	35.43	4.85	9.70
1780	48.37	10.02	30.82	3.25	7.51
1850	19.30	24.10	50.00	.96	5.30
1868	78.00	5.00	17.00	.....	.....
25	38.00	12.00	48.00	.....	2.00
1931	43.00	19.00	35.00	2.00	1.00
Average...	36.79	17.26	38.97	2.40	4.51
S. D.....	±17.91	± 4.28	±12.73	±1.77	±3.22
rs.....	±12.62	± 3.02	± 8.97	±1.21	±2.23
rm.....	36.79±3.63	17.26±0.87	38.97±2.58	2.40±0.35	4.51±0.65

TABLE VI—*Composite differential leucocyte counts of all birds.*

GROUP	POLYMORPHONUCLEARS	LARGE MONONUCLEARS	LYMPHOCYTES	EOSINOPHILES	MAST CELLS
Paralyzed birds with no gross lesions.....	33.28	19.54	40.01	3.90	3.24
Paralyzed birds with gross lesions.....	31.28	17.89	44.12	2.84	3.64
Paralyzed birds with tumors...	36.34	18.58	37.24	4.18m	3.63
Birds with gray eyes.....	36.79	17.26	38.97	2.40	4.51
Average.....	34.42	18.32	40.08	3.33	3.75
S. D.....	± 2.2	± 0.83	± 2.4	±0.7	±0.47
rs.....	± 1.7	± 0.64	± 1.9	±0.54	±0.36
rm.....	34.42±0.9	18.32±0.3	40.08±0.9	3.33±0.27	3.75±0.19

Table VI, a composite of all classes, does not reveal anything that is not indicated in the individual tables. The standard deviations and probable errors are less, which is of significance when compared with the normal.

Table VII gives the average total blood-counts of normal fowls, according to twelve authors as listed by Burnett,[2] and also the average counts as given by Fenstermacher.[5] The average of these was used as the total blood-counts for normal fowls for checking our data.

TABLE VII—*Total blood-counts of normal fowls.*

AUTHOR	ERYTHROCYTES	LEUCOCYTES
Albertoni and Mazzoni	2,460,000	32,300
Ellermann and Bang	3,000,000	30,000
Goodall	3,200,000	19,000
Hayem	2,400,000	26,300
Launey and Levy-Bruhl	2,670,000	27,500
Hedfeld	4,200,000	24,000
Mack	3,000,000	33,777
Moore	3,600,000	20,081
Schmeisser	3,500,000	50,000
Ward	3,280,000	36,185
Warthin	2,500,000	20,500
Burnett	3,300,000	17,921
Fenstermacher	2,594,188	24,425
Average	3,054,168	27,845
S. D.	± 512,000	± 8,400
rs	± 359,508	± 5,935
rm	3,054,168 ± 99,151	27,845 ± 1,686

The average total erythrocytes in the group recorded in table VIII is normal, while a marked increase in leucocytes is evident.

The average of the total counts of the group recorded in table IX shows less variation from the normal than any of the other groups.

In the group recorded in table X, the average total leucocytes is high as compared with the normal.

Table XI indicates that in this group of birds the total erythrocyte counts are slightly low, while the total leucocyte counts are high. The errors seem quite high but on careful analysis it will be noted that these are not unduly high and compare fairly closely to those in the table for normal birds.

TABLE VIII—*Total blood-counts of paralyzed birds with no gross lesions.*

BIRD	ERYTHROCYTES	LEUCOCYTES
1739	3,720,000	48,600
1743	3,512,000	50,000
1738	4,344,000	46,666
900	3,064,000	33,776
Dr. B	3,584,000	64,000
1752	3,504,000	38,000
1753	3,448,000	43,332
45860	2,784,000	20,666
1939	3,550,000	78,400
1947	2,750,000	31,800
1888	3,688,000	43,666
1889	1,576,000	48,333
1890	2,736,000	20,630
1917	2,900,000	42,950
1922	3,230,000	44,950
1918	2,870,000	46,666
1919	3,010,000	40,555
1933	3,540,000	27,330
1934	3,400,000	45,555
1935	3,780,000	25,555
1937	2,950,000	35,555
1938	2,810,000	57,777
1891	3,456,000	29,111
Average	3,226,347	41,907
S. D	± 421,800	±13,304
rs	± 290,912	± 9,173
rm	3,226,347 ±60,705	41,907 ±2,828

The erythrocytes in the group recorded in table XII are fairly normal in number but a considerable increase in the number of leucocytes is noticed.

The standard deviations and the probable errors are much less than in the composite table of normal birds.

Burnett[2] does not state the method used by the men listed for determining the hemoglobin. Fenstermacher[5] used a Dare hemoglobinometer, which resulted in exactly the same average as that obtained by Burnett.

Since no data were available whereby the coagulation time for blood of normal fowls could be learned we determined this on 12 healthy, year-old fowls. It was found that this time varied so much that it is doubtful if, except in extreme cases, it has much significance.

The average hemoglobin content in the blood of the group recorded in table XV is slightly lower than for normal birds and

the average coagulation time is noticed to be prolonged considerably.

As in the previous group (table XV), the hemoglobin is low and the coagulation time prolonged, for the group recorded in table XVI.

In the group recorded in table XVII, the hemoglobin is markedly low on an average.

Again, in the group recorded in table XVIII, the hemoglobin is low and the coagulation time prolonged.

The low hemoglobin content shown in the average of all birds, composite table XIX, and the average composite coagulation

FIG. 1. Large ovarian tumor, as well as hyperplasia of kidneys and spleen.

time, which is twice that obtained for normal birds, undoubtedly are of significance.

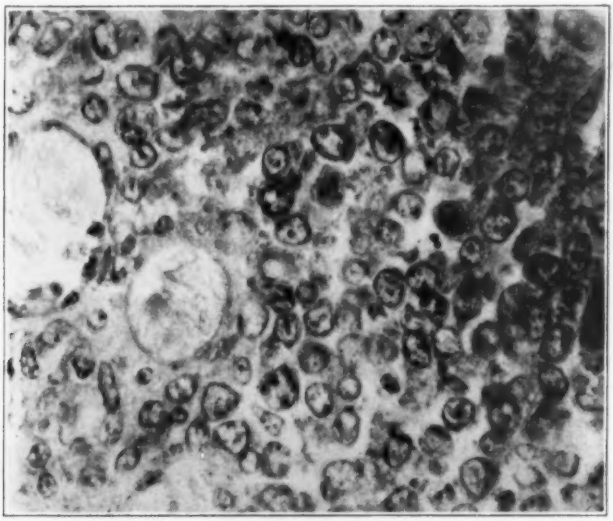

FIG. 2. Photomicrograph of the tumor shown in figure 1.

DISCUSSION AND SUMMARY

In 14 birds of the 31 studied with symptoms of paralysis of the limbs but no gross lesions noticed on autopsy, budding of lymphocytes occurred. In 10 of the 14 studied with symptoms of paralysis of the limbs and lymphatic hyperplasia of visceral organs, budding of lymphocytes was marked. In only three of the 15 paralyzed birds with tumors found on autopsy was budding of lymphocytes noticed. Three of the 12 birds with iritis or gray eyes also had numerous budding lymphocytes.

The blood-smear of one bird with gray eyes revealed vacuoles in the cytoplasm of the large lymphocytes in addition to budding.

In two birds with lymphatic hyperplasia of visceral organs, neutrophile granules were present in the cytoplasm of several of the large mononuclears.

Histologic sections made from the nerves and visceral organs gave evidence of a preponderance of small lymphoid-like cells similar to those described by Johnson[6] in a recent publication.

From the data presented in this paper we notice that, in all the groups of birds studied, the large mononuclears are high as

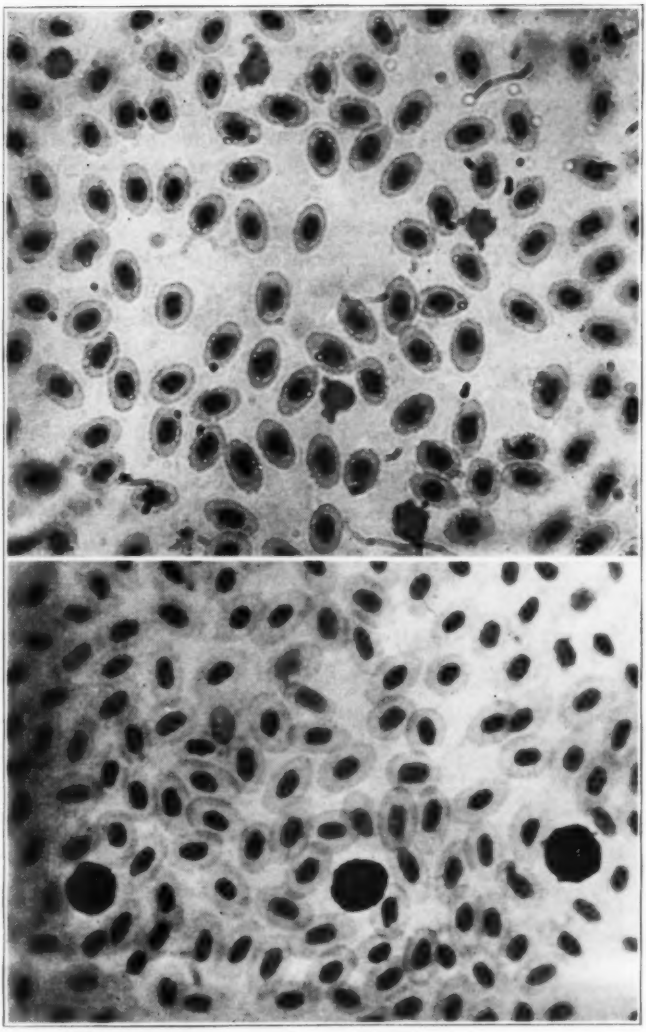

FIG. 3 (above). Budding of lymphocytes.
FIG. 4 (below). Blood-smear from a paralyzed bird with three mast cells in one microscopic field.

compared with the established normal counts. This increase is noticed also in the mast cells. The total leucocyte counts also are high but the total erythrocyte counts are well within the range established for normal birds. The hemoglobin content is low as compared with that of normal birds. The coagulation time is prolonged in most instances, so that the average time is twice that for normal birds although the variation in supposedly normal birds is so great that the importance of this difference would have to be determined by the use of a large number of birds.

The significance of the apparent increase in large mononuclears and mast cells has not been determined in fowls. The increase in total leucocytes associated with budding of numerous lymphocytes would indicate the onset of lymphatic leukemia. The low

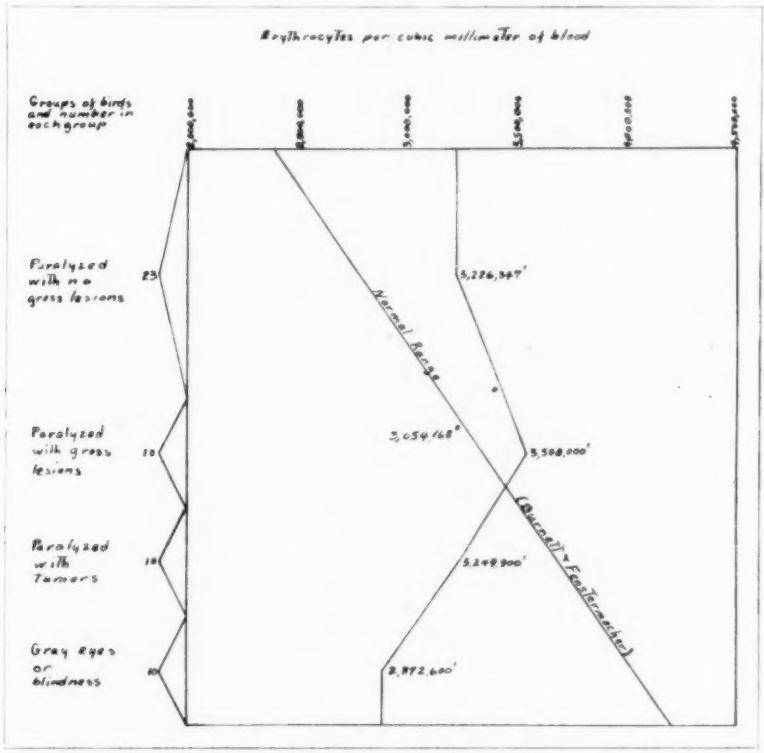

FIG. 5. Graph showing erythrocyte counts in 53 fowls affected with various forms of lymphomatosis. This graph indicates that the erythrocyte range for the four groups of birds is within the established normal range.

percentage of hemoglobin content as well as prolonged coagulation time also is indicative of this disease.

It is suggested from this study that partial control of these conditions, until more is known about the cause and transmission, might be effected by making total and differential blood-counts in the early stages of the disease. In this way affected birds could be detected and removed from the flock.

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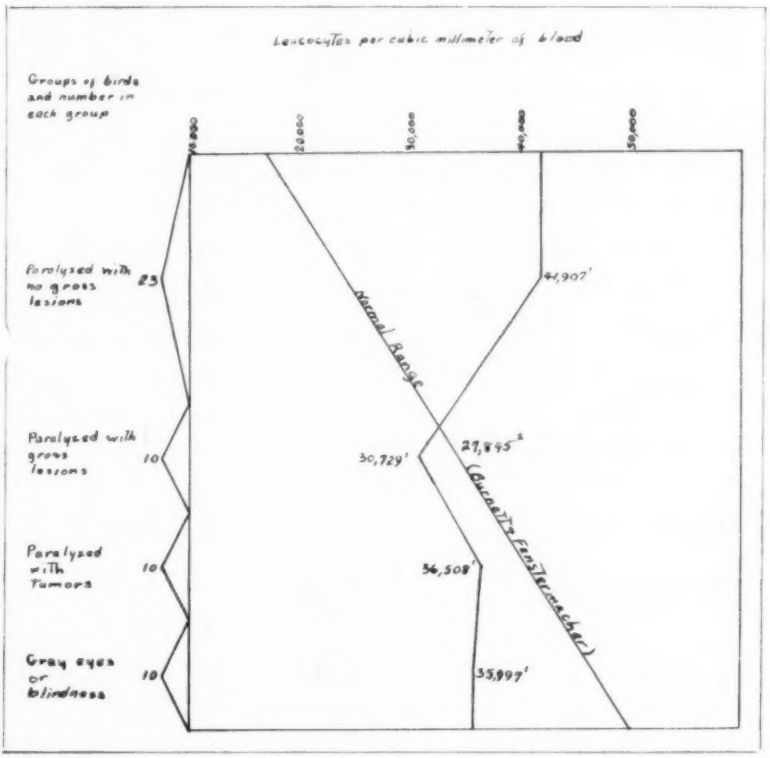

FIG. 6. Graph showing leucocyte counts in 53 fowls affected with various forms of lymphomatosis. This graph is slightly misleading in that the normal extends over to 50,000, which is Schmeisser's count reported by Burnett.[2] This is much higher than any other workers have reported. However, the average normal is not changed greatly and with that in mind it will be noticed that the total leucocyte counts are consistently high.

TABLE IX—*Total blood-counts of paralyzed birds with gross lesions.*

BIRD	ERYTHROCYTES	LEUCOCYTES
1723	3,728,000	33,850
1798	4,504,000	31,776
1799	3,856,000	28,666
H-I	3,832,000	23,110
H-II	3,600,000	21,554
45858	3,392,000	32,000
8	2,552,000	46,333
1893	3,064,000	43,222
1906	4,672,000	26,000
1932	2,280,000	20,777
Average	3,548,000	30,729
S. D.	± 708,000	± 8,000
rs	± 503,117	± 5,733
rm	3,548,000 ± 158,507	30,729 ± 1,821

TABLE X—*Total blood-counts of paralyzed birds with tumors.*

BIRD	ERYTHROCYTES	LEUCOCYTES
1741	2,864,000	49,776
1755	2,520,000	37,110
1782	3,752,000	40,000
1781	4,224,000	35,554
1866	4,189,000	31,111
1867	2,130,000	29,183
1894	3,200,000	33,330
1907	3,368,000	44,222
1915	3,192,000	22,444
1916	3,060,000	42,350
Average	3,249,900	36,508
S. D.	± 638,000	± 7,500
rs	± 451,915	± 5,396
rm	3,249,900 ± 142,994	36,508 ± 1,686

TABLE XI—*Total blood-counts of birds with gray eyes.*

BIRD	ERYTHROCYTES	LEUCOCYTES
1692	2,800,000	33,332
1708	2,632,000	33,776
M-I	3,160,000	38,444
M-II	2,776,000	57,776
90	4,008,000	32,444
1783	2,608,000	29,332
1850	3,720,000	24,000
1868	1,848,000	64,000
25	1,486,000	24,203
1931	3,688,000	22,667
Average	2,872,600	35,997
S. D.	± 764,000	±13,500
rs	± 542,912	±10,117
rm	2,872,600±171,323	35,997±3,035

TABLE XII—*Composite total blood-counts of all birds.*

GROUPS OF BIRDS	ERYTHROCYTES	LEUCOCYTES
Paralyzed birds with no gross lesions	3,226,347	41,907
Paralyzed birds with gross lesions	3,548,000	30,729
Paralyzed birds with tumors	3,249,900	36,508
Birds with gray eyes	2,872,600	35,997
Average	3,224,212	36,285
S. D.	± 2,400	± 3,900
rs	± 1,888	± 3,102
rm	3,224,212±944	36,285±1,551

TABLE XIII—*Percentage of hemoglobin for normal fowls.*

WORKER	HEMOGLOBIN
Ellermann and Bang	57.5
Mack	87.3
Schmeisser	65.0
Burnett	76.0
Fenstermacher	76.0
Average	72.4
S. D.	±10.2
rs	± 7.6
rm	72.4±3.4

TABLE XIV—*Coagulation time for normal fowls.*

BIRD	COAGULATION TIME (MINUTES)	(SECONDS)
1	2	30
2	14	15
3	8	10
4	3	55
5	2	40
6	2	30
7	2	..
8	4	4
9	13	40
10	11	50
11	1	10
12	9	..
Average	6	21

TABLE XV—*Hemoglobin and coagulation time of blood of paralyzed birds with no gross lesions.*

BIRD	HEMOGLOBIN (%)	COAGULATION TIME (MINUTES)	(SECONDS)
B	60	6	30
45860	60	3	30
1888	60	20	..
1889	50	7	..
1890	60	14	..
1891	60	60	..
1906	68	12	..
1917	57	Not determined	
1922	90	8	..
1918	48	3	..
1919	53	3	..
1933	59	8	..
1934	67	15	..
1935	51	15	..
1937	48	30	..
1938	35	Not determined	
1939	52	Not determined	
1947	70	Not determined	
Average	58.2	14	6
S. D	±11.23		
rs	± 7.80		
rm	58.2±1.87		

TABLE XVI—*Hemoglobin and coagulation time of blood of paralyzed birds with gross lesions.*

BIRD	HEMOGLOBIN (%)	COAGULATION TIME (MINUTES)	(SECONDS)
1798	60	7	..
1799	60	6	..
H-I	60	17	..
H-II	60	13	..
45858	60	11	..
1893	50	10	..
1932	40	4	..
Average	55.7	9	42
S. D.	±7.27		
rs	±5.29		
rm	55.7±1.99		

TABLE XVII—*Hemoglobin and coagulation time of blood of paralyzed birds with tumors.*

BIRD	HEMOGLOBIN (%)	COAGULATION TIME (MINUTES)	(SECONDS)
1755	50	9	..
1782	60	11	..
1781	50	2	..
1866	50	15	..
1867	40	14	..
1894	50	..	..
1907	45	5	..
1915	57	20	..
1916	39	10	..
Average	49	10	45
S. D.	±6.54		
rs	±4.71		
rm	49±1.55		

TABLE XVIII—*Hemoglobin and coagulation time of blood of birds with gray eyes.*

BIRD	HEMOGLOBIN (%)	COAGULATION TIME (MINUTES)	(SECONDS)
M-I	50	3	..
90	70	6	..
1783	60	20	..
1850	60	5	..
1868	60	12	..
8	50	1	30
25	37	7	..
1931	62	90	..
Average	56.1	18	4
S. D	±9.44		
rs	±6.80		
rm	56.1±2.40		

TABLE XIX—*Composite of hemoglobin and coagulation time of blood of all birds.*

GROUP	HEMOGLOBIN (%)	COAGULATION TIME (MINUTES)	(SECONDS)
Paralyzed birds with no gross lesions	58.2	14	6
Paralyzed birds with gross lesions	55.7	9	42
Paralyzed birds with tumors	49.0	10	45
Birds with gray eyes and blindness	56.1	18	4
Average	54.75	12	35.5
S. D	±3.45		
rs	±2.70		
rm	54.75±1.34		

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CONVENTION NOTES

Virginia appeared on the registration list eight times. From the Old Dominion those present included: Drs. W. L. Bendix, Dumbarton; Cooper Curtice, Fairfax; George C. Faville, P. T. Galloway, H. C. Givens and A. J. Sipos, Richmond; Page M. Graves, Culpeper; I. D. Wilson, Blacksburg.

The nation's capital contributed nine to the attendance figures. From the Department of Agriculture there were: Drs. Chester N. Dale, C. D. Lowe, W. M. MacKellar, John R. Mohler, H. W. Schoening, J. E. Shillinger and A. E. Wight. From the U. S. Army: Major Seth C. Dildine and Major A. C. Wight.

The meeting attracted nine from *Mississippi*. They were: Drs. Charles B. Cain, State College; Andy Crawford, New Albany; C. D. Crawford, Rolling Fork; E. H. Durr, C. E. O'Neal and R. H. Stewart, Jackson; R. L. Durr, Brookhaven; J. S. Kamper, McComb; M. J. Luster, Clarksdale.

Four of the Canadian provinces had members at the convention. From *Alberta:* Dr. J. C. Phillips, Edmonton. From *Ontario*: Drs. J. A. Campbell, George A. Clark and H. MacDonald, Toronto; H. M. LeGard, Weston; C. D. McGilvray, Guelph; C. A. Mitchell, Ottawa; J. Martin Rice, London. From *Quebec*: Dr. Charles B. Baker, Montreal; M. Gabriel, La Trappe. From *Saskatchewan*: Dr. C. J. Johannes, Saskatoon.

All the way from *California* came fourteen veterinarians to swell the attendance figures. From the Golden State were: Drs. J. M. Arburua, San Francisco; A. G. Boyd and J. A. Lynn, Sacramento; W. H. Boynton, C. H. Haring and F. W. Wood, Berkeley; P. H. Browning, San Jose; L. J. Cook and E. E. Jones, Los Angeles; H. H. Groth, San Mateo; Evelyn Keagy, Beverly Hills; L. O. Lietzman, Whittier; F. H. Saunders, Stockton; R. J. Schermerhorn, Redlands.

THE SENSITIVITY OF CHICKENS TO TUBERCULIN FOLLOWING EXPOSURE TO DIFFERENT VARIETIES OF ACID-FAST BACILLI*

By WILLIAM H. FELDMAN, *Rochester, Minn.*
Division of Experimental Surgery and Pathology
The Mayo Foundation

A series of experiments were done on chickens for the purpose of obtaining information concerning the specificity of the tuberculin reaction and to determine if acid-fast bacteria other than the bacillus of tuberculosis would provoke in them sensitivity to tuberculin.

METHODS OF INVESTIGATION

Fifty-six well-nourished, normal-appearing adult White Leghorn hens, aged approximately ten months, were selected for the experiments, after first being examined for demonstrable physical defects. The wattles of each chicken were simultaneously injected intradermally with both avian and mammalian tuberculin. Approximately 0.02 cc of a 50 per cent solution of Koch's old tuberculin, the usual diagnostic dose for chickens, was given. Tuberculin reactors were never observed in any of the chickens subjected to the preliminary tests.

The chickens given injections with avian, human and bovine forms of *Mycobacterium tuberculosis* were placed in individual cages for the duration of the experiment, while those inoculated with acid-fast bacteria other than *M. tuberculosis* were placed in cages, each accommodating four chickens. Those inoculated with the pathogenic mycobacterium were cared for by a different attendant than those inoculated with the nonpathogenic forms; the attendant caring for the latter had no contact with animals being used for experimental tuberculosis and the chickens were kept in a separate building.

Cultures: The bacillus of tuberculosis was isolated originally from spontaneously infected material. The strains had been typed by pathogenicity tests in guinea pigs, rabbits and chickens, and were characteristically representative of the human, bovine and avian forms of *M. tuberculosis*. Briefly the sources of these cultures were as follows:

Strain 709, avian, isolated in 1928, from the liver of a chicken that died of spontaneous tuberculosis.

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Strain 744, bovine, isolated in 1930, from a bronchial lymph-node of a cow which had reacted positively to tuberculin.

Strain 839, human, isolated in 1930, from the sputum of a human being affected with pulmonary tuberculosis.

Bacterial cultures other than bacillus of tuberculosis of mammalian or avian origin were six in number. These were: *Mycobacterium chelonei; Mycobacterium phlei; Mycobacterium smegmatis,* subcultures obtained in 1931, from Dr. Esmond R. Long, University of Chicago, and *Mycobacterium paratuberculosis;* acid-fast bacteria of soil origin, rough type of growth; acid-fast bacteria of soil origin, smooth type of growth, subcultures obtained in January, 1932, from Dr. William A. Hagan, Cornell University.

Three different varieties of bacteria that had been obtained from the tissues of cattle also were used in an attempt to incite tuberculin sensitivity in chickens. The origins of these were:

Hastings strain GH, subculture obtained from Dr. E. G. Hastings, University of Wisconsin. The organism, which is acid-fast, was isolated in 1927, from the mesenteric lymph-node of a young cow which had not reacted to tuberculin. The organism failed to produce tuberculosis when injected into rabbits, guinea pigs, and chickens.

Daines strain, 42-c-31: The organism was isolated from a so-called skin lesion of a bovine and was considered representative of the organisms classified as group 2 by Daines and Austin.[1] The organism exhibits a tendency toward pleomorphism with diphtheroid forms common. Acid-fastness is a variable feature, being dependent apparently on the constituency of the medium on which it is cultivated and the age of the culture. Daines strain, 24-c-30: This organism also was isolated from a so-called skin lesion of a bovine and was considered characteristic of those organisms belonging to group 3 in Daines and Austin's classification. The bacterium is usually acid-fast regardless of the character of medium on which it is grown. The subcultures were obtained in December, 1931, from Dr. L. L. Daines, University of Utah.

Inoculums: All inoculums were prepared from recently grown subcultures suspended in sterile physiologic sodium chlorid solution. The suspensions purposely were made rather dense and had a turbidity comparable to tube 10 of the MacFarland nephelometer. The respective suspensions were placed in the refrigerator over night, where the larger bacterial clumps were permitted to settle to the bottom of the tubes.

Inoculations: With two exceptions the respective bacterial suspensions were used to inoculate an equal number of chickens intravenously and intratracheally. The amount of bacterial suspension injected into each chicken was 0.5 cc. Of the seven chickens (group 1) inoculated with *M. tuberculosis* of bovine origin, four were given intratracheal injections and three were given intravenous injections. Seven chickens (group 2) were exposed to *M. tuberculosis* of human origin, three intratracheally and three intravenously. Of the six chickens (group 3) inoculated with avian bacillus, two only were inoculated intratracheally, whereas four were inoculated intravenously. The thirty-six chickens in group 4 were divided into nine lots of four each and were given injections of nine different forms of acid-fast bacteria. These included two strains isolated from the soil, *M. phlei, M. chelonei, M. smegmatis, M. paratuberculosis,* two different strains isolated from so-called skin lesions of cattle, and one strain isolated from the mesenteric lymph-nodes of a cow.

Results

The seven chickens constituting group 1 were exposed to the bovine form of *M. tuberculosis.* The results of the various tuberculin tests are recorded in table I.

Table I—*Tuberculin tests of chickens inoculated with the bovine form of Mycobacterium tuberculosis.**

Chicken	Mode of Inoculation	Tuberculin Tests†										Intervals Between Tests (Days)
		1st		2nd		3rd		4th		5th		
		A	M	A	M	A	M	A	M	A	M	
1	Intratracheal	+4	+4	−	−							Chicken killed 23 days after last test; no lesions
2	Intratracheal	−	+4	−	+4	−	+1	−	+1	−	−	49, 44, 52, 30
3	Intratracheal	−	+4	−	−	−	−	−	−	−	−	49, 44, 52, 30
4	Intratracheal	−	+3	−	+2	−	+1	−	+1	−	−	49, 44, 52, 30
5	Intravenous	−	−	−	+4	−	−	−	−	−	−	49, 44, 52, 30
6	Intravenous	−	+4	−	−	−	−	−	−	−	−	49, 44, 52, 30
7	Intravenous	−	+2	+1	+2	−	−	−	−	−	−	117, 77, 30, 30

*Positive reactions to tuberculin are recorded on the basis of +1, +2, +3 and +4, depending on the degree and character of the local tissue reaction. +1 indicates a reaction of the smallest magnitude; +4 of the greatest; the minus sign indicates negative reaction. Chickens 1 to 6 inclusive received the first postinoculation injection of tuberculin 32 days after exposure to the bacterial suspension, and chicken 7 was injected with tuberculin 44 days after being given *Mycobacterium tuberculosis.*

†A is avian tuberculin; M is mammalian tuberculin.

The chickens of group 1, living at the time of the last tuberculin test, were killed for necropsy and in no instance were lesions of tuberculosis observed. The chickens were all in good physical condition at the time of death (fig. 1).

During the period of observation, all of the chickens in group 1 exhibited a sensitivity to mammalian tuberculin and two of them reacted to both avian and mammalian tuberculin (chickens 1 and 7). One of these chickens gave a well-marked reaction to both tuberculins; the reaction of the other chicken to avian tuberculin, although definite, was recorded as +1. It is also evident that although each of the chickens in the series received

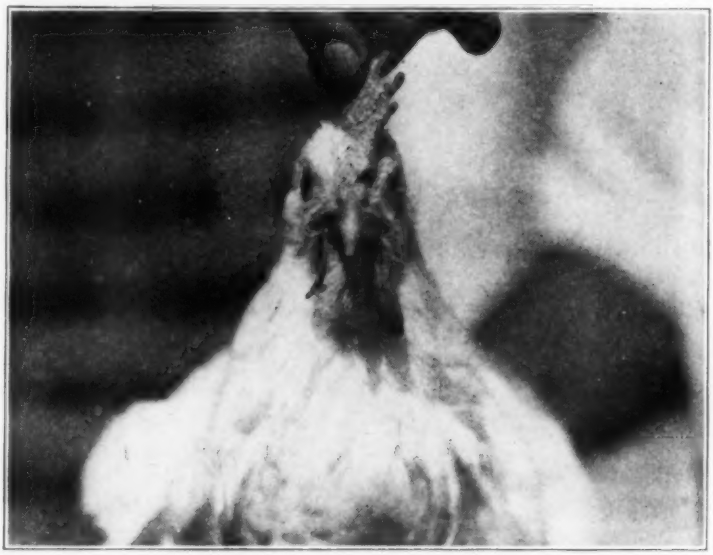

FIG. 1. Chicken 2. Marked reaction to mammalian tuberculin. Intratracheal injection of *Mycobacterium tuberculosis* of bovine origin had been given one month previously. Photograph taken 48 hours after the administration of tuberculin.

the same amount of the same bacterial suspension, considerable variation existed in the susceptibility of the respective chickens as measured by the tuberculin reaction. Although the majority gave a definite reaction to mammalian tuberculin at the time of the first test, one bird (chicken 5) did not show evidence of sensitivity to tuberculin until the second test, or 81 days after injection of the bacteria. Two chickens continued to exhibit a sensitivity for mammalian tuberculin for many months, but in

most of the chickens this phenomenon proved somewhat transitory and was not demonstrable after the second or third test.

It seems significant that of the 16 positive reactions recorded in group 1, only two should have been obtained with avian tuberculin. It may also be noted that occasionally a chicken may be encountered in which reactions of equal magnitude occur with avian and mammalian tuberculin even though the sensitization was the consequence of the bovine form of *M. tuberculosis*. In this group the chickens receiving the bacterial suspension intratracheally seemed to possess a sensitivity for tuberculin for a longer period than those receiving the inoculum intravenously.

The six chickens in group 2 were inoculated with a suspension of *M. tuberculosis* of human origin. The results of the five tuberculin tests are shown in table II.

TABLE II—*The tuberculin tests of chickens inoculated with the human form of Mycobacterium tuberculosis.**

Chicken	Mode of Inoculation	Tuberculin Tests										Intervals Between Tests (Days
		1st		2nd		3rd		4th		5th		
		A	M	A	M	A	M	A	M	A	M	
8	Intratracheal	+2	+4	−	+2							44; animal died 40 days after last test; neoplasia
9	Intratracheal	+4	+4	−	+4	−	+1	−	−	−	−	44, 50, 39, 30
10	Intratracheal	−	+4	−	−	−	−	−	−	−	−	44, 50, 39, 30
11	Intratracheal	+1	+4	−	+2	−	+2	−	−	−	−	44, 50, 39, 30
12	Intravenous	+2	+3	−	+3	−	+4	−	−	−	−	44, 50, 39, 30
13	Intravenous	+2	+4	−	−	−	−	−	−	−	−	44, 50, 39, 30
14	Intravenous	−	−	−	+3	−	−	−	−	−	−	44, 50, 39, 30

*The first postinoculation tuberculin test was administered 52 days after the respective birds received the bacterial suspension.

In all of the chickens in group 2 sensitivity to tuberculin developed following the inoculation of the suspension of the human form of *M. tuberculosis*. Two of the chickens reacted to mammalian tuberculin only, but the four other chickens in the group reacted to both mammalian and avian tuberculin. In most instances, however, the reaction to mammalian tuberculin was greater than that to avian, although in one instance the reaction to both tuberculins was equal (chicken 9, fig. 2).

Positive reactions to both kinds of tuberculin, when evident, occurred at the time of the first postinoculation test. Subsequent tests failed to disclose similar phenomena.

Tuberculin sensitivity was not observed in one chicken until the second test, which was 76 days after it had received the inoculation of bacteria, and all subsequent tests were negative (chicken 15). Chicken 10 manifested +4 sensitivity to mammalian tuberculin 32 days after receiving the bacterial suspension, yet all later attempts to obtain a reaction to tuberculin were futile. The data pertaining to another bird (chicken 11) are also of some interest in that first, second and third tests were positive, the fourth test was negative and finally a +1 positive reaction was observed with the fifth test.

FIG. 2. Chicken 9. Marked reaction to both avian and mammalian tuberculin. The chicken had been inoculated 34 days previously, with the human form of *Mycobacterium tuberculosis*. Tuberculin was administered 48 hours before chicken was photographed.

The six chickens in group 3 were exposed to the avian form of *M. tuberculosis* (table III).

The reactions of the respective chickens in group 3 to tuberculin were erratic, perhaps because of the virulence of the infecting organism. Although a very dilute suspension of bacteria was used for inoculation, it was apparently sufficient in most instances to induce an overwhelming infection. However, a marked tuberculous process did not occur in chicken 15, although it received a comparable dose of the same suspension of *M. tuberculosis* used to infect the others in this group. Chicken

15 died 34 days after the third injection of tuberculin, and at the time of death was in excellent physical condition with the exception of nontuberculous peritonitis and a somewhat abnormal ovary of doubtful significance. Gross lesions of tuberculosis were not apparent, and histologically the only evidence of a possible tuberculous infection consisted of a few atrophic collections of monocytic cells in the liver. Acid-fast bacilli were not discernible in appropriately stained sections. Why a definite tuberculous infection failed to develop in this chicken is inexplicable. The chicken had received the bacterial suspension 120 days before death, which was sufficient time for the disease to become established. The fact that well-marked tuberculous lesions developed in the other chickens that received portions of the same inoculum makes it difficult to understand why the organisms failed to produce tuberculosis in chicken 15.

TABLE III—*Tuberculin tests of chickens inoculated with the avian form of Mycobacterium tuberculosis.**

CHICKEN	MODE OF INOCULATION	TUBERCULIN TESTS										INTERVALS BETWEEN TESTS (DAYS)
		1ST		2ND		3RD		4TH		5TH		
		A	M	A	M	A	M	A	M	A	M	
15	Intratracheal	−	−	+3	−	+3	−	+1	−			28, 28, 30; died 34 days after last test; peritonitis
16	Intratracheal	−	−	−	−	+2	+2					28, 28; died 26 days after last test; tuberculosis
17	Intravenous	+3	−	+3	−							28; died 1 day after last test; tuberculosis
18	Intravenous	+4	+3	−	−	−	−	−	−	−	−	28, 28, 30, 63; killed 22 days after last test; tuberculosis
19	Intravenous	+2	−	−	−							30; killed 16 days after last test; tuberculosis
20	Intravenous	−	−	+1	−	+1	+1					30, 31, 60; died 12 days after last test; tuberculosis

*All chickens, with the exception of chickens 19 and 20, were given the first postinoculation tuberculin test 28 days after receiving the bacterial suspension. Chickens 19 and 20 were injected with tuberculin 14 days after receiving the bacterial suspension.

All of the chickens were sensitive to avian tuberculin, although three were sensitive but once during the period of observation. Three of the chickens manifested sensitivity to both avian and mammalian tuberculin, and the two reactions were approximately of the same magnitude in the respective chickens.

A lack of correlation between the character or degree of the tuberculous infection and the sensitivity to tuberculin was a notable feature of some of the chickens in this group. Chicken 18 reacted positively to both avian and mammalian tuberculin 28 days after receiving inoculum. All subsequent attempts to demonstrate sensitivity to tuberculin were futile, although a tuberculous process was found to be well established at necropsy 199 days after exposure to *M. tuberculosis*.

Since all chickens in group 3 received similar amounts of the same infective agent, it is evident that the degree of susceptibility and resistance varied considerably with the respective chickens.

The 36 chickens constituting group 4 were inoculated with acid-fast bacteria other than *M. tuberculosis*. The chickens were subjected to five separate tuberculin tests over a period of approximately six months. The first test was administered 30 days after the respective chickens had received the bacterial suspension. Both avian and mammalian tuberculin were injected simultaneously.

Twenty-two of the chickens were living when the experiment was terminated, seven months after it was begun. Fourteen had died of various causes after being under observation for from two to 174 days. The cause of death of ten of the 14 chickens was as follows: leukosis, two; fatal injuries as a consequence of attacks by cage mates, five; neoplasis, one; pyogenic pulmonary abscess one, and egg-bound, one. The cause of death of four of the chickens was not apparent.

With the exception of three of the four chickens which were inoculated with the suspension prepared from the Hastings strain of acid-fast bacteria, sensitivity to either avian or mammalian tuberculin was not observed in the chickens in group 4. Neither was there morphologic evidence of infection, as a consequence of the injections of the various bacterial suspensions when the respective chickens were examined at necropsy.

The three chickens in the series inoculated with the Hastings strain of acid-fast bacilli and which were shown subsequently to be sensitive to tuberculin were also devoid of demonstrable lesions at necropsy. The results of the first tuberculin test of

these three chickens showed that one chicken reacted +4 to avian tuberculin and +1 to mammalian tuberculin (fig. 3). The other two chickens failed to react to the first test. At the time of the second test, administered 26 days after the first, the chicken that reacted previously to both avian and mammalian tuberculin failed to react positively. One of the other chickens gave a +3 reaction to mammalian tuberculin at the second test, but was negative to avian tuberculin. The third chicken failed to react to avian tuberculin at the second test but gave a +1 reaction to mammalian tuberculin at this time. Each of these three chickens was negative to both tuberculins on each of three subsequent tests.

FIG. 3. Tuberculin sensitivity in a chicken given intratracheal injection 32 days previously, with Hastings strain of acidfast bacillus. Right wattle injected with avian tuberculin; reaction, +4. Left wattle injected with mammalian tuberculin; reaction, +1. Photograph taken 48 hours after tuberculins had been administered.

Attempts were made to demonstrate the pathogenicity of the Hastings strain of acid-fast bacteria by injecting into four guinea pigs and four rabbits suspensions of the bacteria in physiologic sodium chlorid solution. Two of the guinea pigs died within a few days; one death was due to pneumonia, the cause of the other death was not apparent. The other two guinea pigs did not have significant lesions when killed eight weeks

after injection. Three of the rabbits died three weeks after intravenous exposure to the bacterial inoculum without significant lesions. The remaining rabbit was killed after a lapse of eight weeks and was found to be free of disease.

Although the evidence is not entirely conclusive, pathogenicity for the Hastings strain of acid-fast bacilli has not been demonstrated by the occurrence of demonstrable tissue alterations in guinea pigs, rabbits and chickens inoculated with generous amounts of the organism. I am not convinced, however, that this organism is entirely innocuous, since the well-defined reactions of three of the chickens following the administration of tuberculin would suggest that the organism possessed at least limited pathogenicity. Failure to confirm this by the presence of lesions in the chickens may have been due to their recovery from the infection and the subsequent disappearance of lesions. This particular organism should be studied further to determine if possible its relationship to *M. tuberculosis*.

Briefly summarized the results of this study show that of 20 chickens inoculated with virulent strains of *M. tuberculosis* of human, bovine and avian origin, all revealed demonstrable sensitivity to either avian or mammalian tuberculin, and ten of the series reacted simultaneously to both tuberculins. Of the 36 chickens injected with acid-fast bacteria (other than *M. tuberculosis* of warm-blooded animals) only three reacted positively to tuberculin and none of these possessed significant lesions at necropsy. The three chickens belonged to a series of four inoculated with a bacterial suspension prepared from a strain of acid-fast bacilli obtained from Dr. E. G. Hastings, of the University of Wisconsin, the identity of which has not been established.

COMMENT

Although tuberculin must be considered a dependable agent in revealing the presence of tuberculous infections, the work of Dolgopol[2] has shown definitely that a positive reaction in mammals following the administration of avian tuberculin does not necessarily indicate infection by the avian form of *M. tuberculosis*. The possibility of the occurrence of a group reaction between different forms of pathogenic mycobacteria must be considered in interpreting the tuberculin test, although there is inconclusive evidence that acid-fast bacteria other than *M. tuberculosis* will provoke a demonstrable sensitivity to tuberculin in cattle, swine or chickens.

Crawford[3,4] reported that guinea pigs may be sensitized to both avian and mammalian tuberculin by various acid-fast bac-

teria such as *M. phlei, M. chelonei, M. smegmatis,* "milk bacillus," "fish bacillus," and "mist bacillus." Although none of the guinea pigs inoculated with the microörganisms mentioned exhibited a systemic reaction to either mammalian or avian tuberculin, the skin revealed a definite sensitization to both forms of tuberculin, whereas the mammalian variety provoked a greater number of positive reactions than was obtained by the avian variety. Long[5] also demonstrated the existence of a group relationship between different acid-fast bacteria by obtaining a definite testicular reaction when the "frog," "grass" and smegma bacilli are injected into the testes of tuberculous guinea pigs.

Hagan and Zeissig[6] have shown the existence of a reciprocal relationship between avian tuberculin and johnin in infections caused by *M. paratuberculosis.* They demonstrated also that spontaneously infected tuberculous chickens in a large percentage of cases will react positively to johnin injected intradermally into the wattle and that the reactions obtained were not inferior to those obtained with avian tuberculin. The observations of Hagan and Zeissig[6] have provided sufficient data to justify the use of avian tuberculin in the clinical diagnosis of paratuberculosis of cattle.

Hastings, Beach and their collaborators[7, 8] have advanced the hypothesis that nonpathogenic forms of the genus Mycobacterium may spontaneously sensitize cattle to tuberculin. They were able to demonstrate experimentally a transitory and somewhat inconsistent sensitization in a group of cattle given injections of what were considered to be nonpathogenic acid-fast bacilli isolated from the tissues of so-called no-lesion tuberculin-reacting cattle. Hagan,[9] however, using much smaller amounts of saprophytic acid-fast bacilli, including some cultures obtained from Hastings and Beach, failed in most instances to detect sensitization following the administration of tuberculin. Hagan also mentioned the difficulty of eliciting in cattle a sensitivity to tuberculin by drenching the animals with large amounts of saprophytic acid-fast bacilli obtained from the mesenteric lymph-nodes of apparently normal cattle. Crawford also failed to provoke sensitivity to tuberculin in cattle following their repeated exposures to *M. phlei,* the mist bacillus and the hog skin bacillus.

That cattle may become sensitized to tuberculin through contact with the organism of avian tuberculosis was pointed out by Schalk,[10] and more recently by Marsh, Warren and Morrow.[11] Although capable of sensitizing cattle to tuberculin, it is unusual for the avian form of *M. tuberculosis* to incite morphologic evidence of its presence in this animal. The fact that the sen-

sitization to tuberculin is transitory would indicate the inability of the avian bacillus to prosper in the tissues of cattle.

Apparently, although it seems to be possible to induce a tuberculin sensitivity in guinea pigs by inoculating them with various saprophytic acid-fast bacilli, a similar phenomenon is produced experimentally in cattle with much difficulty if at all. Chickens likewise are usually incapable of sensitivity to either avian or mammalian tuberculin when exposed to other than *M. tuberculosis* of warm-blooded animals. These facts lead one seriously to doubt if any appreciable percentage of cattle which react to tuberculin do so because of a group sensitivity provoked by nonpathogenic species of the genus Mycobacterium. It is conceivable that this might occasionally occur, but until data to the contrary are more convincing it seems more logical to explain the tuberculin sensitivity on the basis of an infection by one of the forms of Mycobacterium responsible for tuberculosis in cattle, chickens and human beings.

A word should be said concerning the dosage of the tuberculin used. The amount injected equalled approximately 0.01 cc of concentrated tuberculin. This is, of course, an exceedingly large dose for so small an animal as a chicken, when compared with the amount usually used for cattle or for human beings. This dosage was selected because it is comparable to the amount of avian tuberculin generally used in detecting spontaneous tuberculous infection in chickens by the intradermic test, after the method was described and recommended by Van Es and Schalk.[12] The test appears to be remarkably reliable, particularly in its failure to elicit false positive reactions in nontuberculous chickens. Apparently as far as chickens are concerned, it is rather highly specific in indicating exposure of the animal to a pathogenic species of Mycobacterium. With the exception of three chickens injected with the Hastings strain of acid-fast bacilli, the large amount of tuberculin injected failed to demonstrate the existence of a sensitivity in chickens inoculated with acid-fast bacteria other than the human, bovine and avian forms of *M. tuberculosis*. Whether similar results would have been obtained had lesser concentrations of tuberculin been used is problematic, although the few observations I have made on this phase of the question indicate that the accuracy of the test is not impugned by diminishing markedly the concentration of tuberculin injected.

SUMMARY

A study was undertaken to determine the capacity of certain pathogenic and saprophytic acid-fast bacteria to sensitize chick-

ens to avian and mammalian tuberculin and to obtain information relative to the specificity of the two forms of tuberculin in indicating if a given chicken had been exposed to a mammalian strain of *M. tuberculosis* or to the avian form of the microörganism. Fifty-six chickens were selected and all were considered free of tuberculosis by virtue of their failure to give a positive reaction to both avian and mammalian tuberculin in doses containing approximately 0.01 cc of the concentrated product. The chickens were divided into four groups: group 1, in which *M. tuberculosis* of bovine origin was given; group 2, in which the human form of the organism was given; group 3, in which the organism of avian tuberculosis was inoculated, and group 4, in which 36 chickens divided into nine lots of four each were given injections of nine different forms of acid-fast bacteria.

The inoculations were made intravenously and intratracheally and the chickens subsequently were subjected to five different tuberculin tests, avian and mammalian tuberculin being injected in opposite wattles simultaneously.

Of the chickens receiving the bovine and human forms of *M. tuberculosis*, more exhibited sensitivity to mammalian tuberculin than to avian tuberculin, although of the 14 chickens in these two groups, none of which revealed lesions of tuberculosis, seven reacted to both tuberculins. Three of the six chickens affected with the avian form of tuberculosis gave a positive reaction to mammalian tuberculin besides the reaction obtained as a consequence of the avian tuberculin.

With the exception of three of the four chickens inoculated with the acid-fast organism obtained from a mesenteric lymph-node of the cow, none of the animals in group 4 revealed the slightest sensitivity to either avian or mammalian tuberculin.

Conclusions

1. Chickens, although not considered susceptible to infection by the bovine or human form of *Mycobacterium tuberculosis*, may become definitely sensitive to mammalian tuberculin and in some instances to avian tuberculin following exposure to these species of Mycobacterium. The sensitivity is transitory.

2. A group relationship exists between the forms of Mycobacterium responsible for tuberculosis in warm-blooded animals, and tuberculin prepared from the avian and mammalian bacilli of tuberculosis.

3. Eight strains of acid-fast bacteria other than those responsible for tuberculosis in cattle, human beings and chickens

were of no significance in eliciting sensitization in chickens to either avian or mammalian tuberculin. As a prerequisite for the development of sensitivity to tuberculin, it seems essential for the animal to experience contact with a pathogenic form of Mycobacterium.

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CONVENTION NOTES

Eight *Kentuckians* signed the register: Drs. E. P. Farley, Paducah; Harry Gieskemeyer, Newport; Charles E. Hagyard, Lexington; G. M. Horton, Mount Sterling; F. H. Riester, Buechel; F. O. Schneider, Nicholasville; C. E. Stockton, Versailles; J. B. Way, Louisville.

Kansas sent twenty-eight of her favorite sons: Drs. J. A. Bogue, Wichita; R. W. Boone, De Soto; Charles W. Bower, Topeka; R. F. Coffey, Eskridge; B. W. Conrad, Sabetha; Major C. E. Cook, Fort Leavenworth; Drs. E. R. Davis, Ottawa; W. M. Dicke, Paola; R. R. Dykstra, E. R. Frank, E. J. Frick, William E. Jennings, F. B. Jones, E. E. Leasure, J. P. Scott and Major Harry E. Van Tuyl, Manhattan; Drs. L. A. Hammers, Clearwater; M. J. Hammond, Broughton; V. C. Hurtig, Courtland; C. B. Kern, Beloit; T. J. Leasure, Lawrence; Col. J. A. McKinnon, Fort Riley; Drs. John Pugh, Phillipsburg; H. E. Schaulis, Clay Center; M. P. Schlaegel, Burr Oak; N. D. Stanley, Hope; J. F. Thomas, Oswego; W. W. Wiseman, Delphos.

EQUINE TRYPANOSOMIASIS-"MURRINA" OR "DERRENGADERA"

Some Notes on the Disease in Panama*

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The first scientific record made in regard to the presence of equine trypanosomiasis (murrina or derrengadera) on the isthmus of Panama was written in 1910, by Darling.[1] He established the fact that it was a trypanosomiasis and named the pathogenic agent *Trypanosoma hippicum*. The typical parasite, according to Darling, is 16 to 18μ in length and 2μ wide. The distance from the kinetoplast to the posterior tip is 1.75μ and the distance from the posterior tip to the middle of the nucleus is 7.5 to 10μ. The flagellum is usually short and frequently not entirely free, for often the attenuated process of the cytoplasm extends to the extreme end of the chromatin filament. At times there is a free flagellum reaching 4μ in length. The posterior tip of this trypanosome is rather blunt. The cytoplasm usually contains scattered basophilic granules and the majority of these granules are in the anterior half of the parasite. A well-developed undulating membrane is present.

It is well known that there may be variations in the morphology of trypanosomes especially during the rapid development of a newly acquired infection and also in the very late stage of the chronic infection in resistant animals. Darling[2] believed it quite evident that this parasite was different from *T. equinum* of mal de caderas and *T. equiperdum* of dourine. Laveran[2] expressed the opinion that *T. hippicum* differed from those just mentioned since it possessed a centrosome, basophilic granulations, and a short flagellum. Wenyon[3] believes that *T. hippicum* is quite similar to *T. venezuelense* and *T. evansi*. We think now that he may be justified in holding this view. The clinical picture of this disease of solipeds had been known, before Darling's report on the subject, as "murrina" or "derrengadera" by the people of Panama. It has always been considered as uniformly

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fatal to horses and mules. The course of illness ranged from a few weeks to several months. According to our personal experience it runs a shorter course in the horse than in the mule. The burro is the soliped with the greatest resistance to the disease. This little animal seldom acquires it but when infected it becomes an important reservoir for the trypanosome over a long period of time.

Clinical Diagnosis

The first cases that appear in a herd are apt to escape attention since it requires a period of a few weeks for the disease to develop significant clinical evidence and a few months for it to gain an extensive spread in a herd of animals. The spread of the disease is far more rapid in herds on pasture than in a herd kept in stables.

The clinical picture is exhibited by fever of an irregular character, progressive emaciation, anemia, faint icterus, rough coat and sometimes edema of the most dependent part of the abdominal wall and sheath and to a less extent of the legs. Late in the illness there is a marked weakness of the posterior extremities and the animal walks with a stiff, staggering gait, frequently dragging the hoof. There is no impairment of appetite throughout the course of the disease. An emaciated animal with fever, a staggering gait, and a good appetite is strong presumptive evidence of this disease and laboratory tests are indicated. These may consist of blood-film examinations for the detection of trypanosomes, inoculations of susceptible animals and the application of the complement-fixation test for equine trypanosomiasis. We select the thick-blood-film method of Barber and Komp[4] for rapid field surveys, since it is difficult to examine fresh blood-films in the field in cloudy, rainy weather. A herd should be examined every two or three days for at least ten surveys in order to pick out the infected cases and, when possible, it is well to apply the complement-fixation test, as recommended by Mohler[5] for dourine. Animal inoculations using 2 to 4 cc of blood by the intraperitoneal method may reveal, at times, a case that these methods have missed but the combination of the thick-blood-film and complement-fixation tests at intervals will soon make it possible to segregate all of the positive animals.

Pathology

Darling[2] has recorded the pathological anatomy in his report but we consider it difficult to establish a diagnosis safely at autopsy unless trypanosomes can be found in the blood or tissue

films made at the time of autopsy. The lesions described for the disease afford supportive evidence of great value but no gross lesions are absolutely characteristic of this disease in the absence of films positive for the trypanosome.

Mode of Transmission

Dourine[5] is described as a disease of breeding animals and is transmitted from animal to animal by coition, while other means of transmission are considered so rare that they have no practical importance in measures for suppressing the disease. Murrina, our local equine trypanosomiasis, can be transmitted by coition but it differs from dourine in being spread also by other means. Any mechanical means that can apply fresh wet blood in a fair amount from an infected animal to an open moist wound in the skin or mucous membranes of another animal may transfer the disease. It can pass through the normal mucosa of the mouth, vaginal vault, etc., if heavily infected blood can be introduced and retained. In our opinion, an important mechanical means of transfer is caused by the crowding and rubbing together of animals under excitement such as being driven in from pastures and caught up in corrals. Animals in this region are covered with innumerable tiny tick and fly bites, thorn and wire injuries, bat bites, harness and saddle galls or rope burns that ooze. Such animals, when they contract the disease, can transfer it by brushing against the sides of negative animals that contain many oozing small wounds. Insects may carry infected fresh wet blood to harness and saddle galls, rope burns, etc., on uninfected members of a herd.

A breeding herd consisting of horses, mules and burros did not show a limitation of the disease to brood mares and stallions. Four colts from eight to ten months of age and two mule colts of the same age were victims of this disease. Nine young geldings and twelve young mares as well as six old mules contracted the disorder. The two stallions belonging to this herd were kept in stables while the other animals were kept in four separate pastures. It is interesting to note, however, that 30 out of 47 old brood mares and one of the two stallions had the disease.

The Supply Department of the Panama Canal, a few years ago, purchased 127 brood mares for the Miraflores Farm from a ranch near Divalá in the province of Chiriqui, Republic of Panama, but this disease has never appeared there according to histories given us by local owners and our recent survey of that region (858 horses and mules), as well as two former

surveys, failed to locate a case of the disease. Thirty of these brood mares acquired the disease on the Miraflores Farm. The stallion found positive at Miraflores was imported from France via Venezuela.

The Vampire Bat as a Vector of the Disease

Dunn,[6] in 1932, discovered from his experimental work on some local species of bats that our vampire bat, *Desmodus rotundus murinus* Wagner, could acquire the disease by feeding on infected animals. He also obtained positive results in five out of six experiments in the transmission of *T. hippicum* to horses, mules and guinea pigs through the bites or feedings of these infected vampire bats. The incubation period in this bat is from six to eight days. The disease proves fatal to the bats in from nine to 27 days after their blood-films reveal the trypanosome. The appetite of the bat is not impaired by the disease and since the animals feed every night and live usually for about a month after becoming infective, there is ample opportunity for them to spread the disease. One of these bats, if unmolested, will feed from one to two hours, voiding urine at frequent intervals. They will average 16 cc of defibrinated blood at a meal if deprived of their natural blood-meal from live animals. This should make it possible for them to infect themselves from relatively light carriers of the parasite. Fortunately, protection from bats can be effected with greater ease than is the case with diptera or other arthropods. Adequate protection can be provided by illuminating the stables either by electricity or by clean oil-lanterns. The owner of a large herd of range animals that cannot be stabled over night is confronted with the more serious problem of keeping the index of infected animals as low as possible to avoid infection of the local vampire bats.

The Question of Insect Vectors

We know that flies can transmit this trypanosome by mechanical means and that blood-meals secured from infected animals by flies and removed from the alimentary tract of the insects within a short time after feeding can be injected into animals and produce the disease but it is still an unsolved question whether some one or more of the horse flies common to this locality may be a host for a generative stage of the parasite.

Jennings[2] believed at the beginning of an epizoötic that the infection might gain its foothold through the bite of a tabanid fly and that very likely only one species was involved. Since

the days of Darling and Jennings, we have learned that many species of the tabanid flies are common to the Isthmus and they breed and range through forested as well as unforested regions. Some circumstances connected with the history of the disease indicate that these flies are open to suspicion as vectors but proof will be hard to secure since these insects, in our experience cannot be kept in captivity for a period long enough to study the question. Studies are in progress with ticks, mosquitoes, flies and *Triatoma geniculata*, but up to the present there is nothing of note to report. The important local epizoötics that we have observed since 1909 have always been confined to the coastal plains or the swampy regions along lakes and rivers where several of the tabanid flies are known to breed.

Animals Susceptible to the Disease

A special report[7] by Clark and Dunn contains the details in regard to this subject but it may be stated briefly that all of our local wild and domestic animals as well as laboratory animals can be experimentally infected and they usually die in a few weeks. Animals infected that lived many months are as follows:

The small native mule (3), from 10 to 16 months.

The domestic cat, 1 year and 12 days.

The white tailed deer, *Odocoileus chiriquensis* Allen, 4 months.

The brocket deer, *Magama sartorii reperticia* Goldman, 6 months.

The collared peccary, *Pecari angulatus bangsi* Goldman, 10 months.

Animals infected but which recovered spontaneously are as follows:

Goat (half grown): Negative for the infection in a very few days.

Sheep (adult): Became negative in about 7 months.

Hog (48 lbs.—domestic): Became negative in about 2 months.

Calf (8 months old): Became negative to guinea pig tests in about 4 months.

Chickens could not be infected.

Iturbe[8] states that in South America spontaneous infections have been found in the capybara, the domestic dog, the fox and certain species of monkeys but this has not been the case with our species of capybara, the dog or our local species of monkeys.

The only animals, aside from the solipeds, that we have ever found carrying a spontaneous infection of *T. hippicum* were cattle. A herd of cattle ranging with a herd of horses and

mules that were attacked by the disease failed to show the presence of *T. hippicum* in blood-films but by inoculations of 2 to 4 cc of blood from the jugular vein into guinea pigs it developed that 4.5 per cent of the cattle were carrying the parasite without harmful results. Our calf experiment indicates that such cattle may be infective to guinea pigs for four months, thus the dangerous bat reservoirs, next in importance to the solipeds, are cattle. Where cattle and horses range together without protection at night from vampire bats, the question of control of this form of trypanosomiasis becomes a serious problem. We think it safe to assume that the animal reservoirs of greatest importance for the perpetuation of *T. hippicum* in our locality are, in the order named, the mule, horse and cattle. Those of secondary importance are the wild and domestic hogs, deer, sheep, goat and domestic cat.

Immunity

Infected guinea pigs give birth to negative offspring and these offspring can be given the disease artificially and killed by it in the usual period of time. During the past 18 months, it has been possible to follow the dams and foals in eight instances and the colts were all negative even though six of the dams were positive. Including colts up to one year of age and their dams, it is possible to list 27 with the following results noted during the observation period of the epidemic:

Dams positive and colts negative	14
Dams negative and colts positive	9
Dams and colts, both positive	4

One horse in the advanced stage of the disease was treated and cured. Some months later it was experimentally reinfected and died of the disease.

Variation in Strains of T. Hippicum

Guinea pigs inoculated from different horses in the same epizoötic do not always behave the same. Some guinea pigs will live many months while others live but a few weeks. When such strains are passed from pig to pig for several months, the average life of the pigs is found to be about the same. One of our strains (colt 22) has killed 33 pigs in an average time of 31 days, yet the range was less than a month up to five months. A second strain (mare 31) has killed 36 pigs in an average time of 37 days and with a range of less than a month up to five months.

Local Records of the Disease

The first established record of a case was of course made by Darling,[1] in 1909, but the clinical character of the disease and losses due to it seem to have been known long before this date. The records passed down through one old family that has always been engaged here in stock-raising indicates that epizoötics of this character did not occur in Panama until large business contacts with other nations started the importation of stock. Whatever may be the facts regarding introductions from outside, it is certain that the disease has been enzoötic in the southern half of Panama for a very long period of time. Outbreaks of the disease have not appeared in Panama during the last 22 years with seasonal regularity, yet most of the widespread epizoötics are said by local stock-owners to have made their appearance in the months from September to January. There is usually a period of a few years between large epizoötics. We believe this may be due in part to the slow replacement of animals killed by an epizoötic, thus greatly reducing the number of contacts or exposures. Epizoötics of major or minor degree, in our experience, have occurred in every month of the year.

Recent Regional Surveys

We found this disease in the stock-breeding farm of the Panama Canal at Miraflores, on December 29, 1929. This farm and the stables border the west side of Miraflores Lake. It is said that eight deaths had occurred in the herd from October to December, 1929. Immediate steps were taken to conduct a blood-film survey of all Canal Zone horses, mules and burros as well as available animals as far down the Pacific coast as Chepo and as far up the coast as Aguadulce and Las Tablas. An area on the Atlantic coast and another on the Pacific coast lying along the boundary of Costa Rica also were examined. (See table I.)

During the year 1930, the main focus of infection on the Pacific side was at Miraflores and it extended a few miles up the coast as far as Venado Beach and Bruja Point. A second focus was found on the Atlantic end at and below the Gatun Dam and along the Chagres River below the Spillway. This last named focus spread during 1931 along the west bank of Gatun Lake and perhaps caused in addition the four cases at Fort Davis on the east side of the dam. The Miraflores focus extended to the Chiva Chiva Trail area across the canal but did not invade the Army herds at Fort Clayton and Corozal Post. Two

cases were found (1931) in the center of the Canal Zone, at Summit, which is also on the east side of the canal. At the close of the year 1931, and during the first half of 1932, a focus appeared about Pacora and Chepo.

The Panama Canal placed a quarantine on the Miraflores and Gatun Dam foci. The herd at Miraflores Stock Farm was kept in quarantine pastures and was subjected to treatment after a few of the old and useless members of the herd had been destroyed. This disease always has been considered fatal and it has been the custom since 1910 either to kill or isolate in screened quarters every case of the disease found in the Panama Canal Zone.

TABLE I—*Results of surveys, 1930 and 1931.*

REGION OR ORGANIZATION	1930		1931	
	EXAM.	POS.	EXAM.	POS.
United States Army, Panama Canal Dept.	1,840	1	1,876	4
Pacific third of Panama Canal Zone	241	64	109	3
Central third of Panama Canal Zone	94	1	207	2
Atlantic third of Panama Canal Zone	157	13	65	1
West bank Gatun Lake	231	9	343	26
East bank Gatun Lake	7	0	67	0
Panama City—Sabans to Chepo, R. de P.	657	0	167	0
Colon-Cristobal	176	0	0	0
Paja to Aguadulce & Las Tablas, R. de P.	251	1	419	0
Almirante, R. de P., United Fruit Co	29	0	0	0
Puerto Armuelles, R. de P., U. F. Co	157	0	858	0
Totals	3,840	89	4,111	36

All herds were slowly decimated wherever the disease made its appearance. New trypanocidal drugs have been manufactured during the last ten or fifteen years for the treatment of human and animal forms of trypanosomiasis but no extended observations had been made on the use of these drugs against *T. hippicum*. The Miraflores herd was sufficiently valuable to warrant an effort to salvage the sick animals and to attempt to control the spread of the disease in the quarantined animals by the use of curative and prophylactic treatments. All animals found positive by repeated blood-film surveys in the other areas mentioned were destroyed, except in two areas to be discussed later. All past local experience with preparations of mercury, arsenic and antimony had not been satisfactory. Tartar emetic seemed to be the only drug that would kill *T. hippicum* but unfortunately an effective course of treatment with this drug was

extremely toxic and usually proved fatal. At the time this epizoötic first was recognized, none of the new trypanocidal remedies were available on the Isthmus in sufficient quantity for the treatment of the herd, so we collected the local supply of tryparsamide and Bayer 205 (naganol) and started treatment in this herd of brood mares, foals, juveniles, stallions, mules and burros. Later a large supply of Bayer 205 was secured. This non-metalliferous remedy was given with tartar emetic during most of the treatment period. The following is a brief account of the treatment administered by Dr. T. L. Casserly:

> The entire herd was first treated on January 9, 1930, using Bayer 205 intravenously. The drug was dissolved in distilled water and the dosage used was 4 grams for mature animals, 2 grams for colts and small animals.
>
> The second treatment was given on January 16, 1930, using tryparsamide in 5-gram doses.
>
> The third treatment was given on January 23, 1930, employing Bayer 205, averaging 3 grams to mature animals and 1.5 grams to colts and small animals.
>
> From February 9, 1930, to April 1, 1930, several animals whose blood-films still showed trypanosomes as well as several that showed clinical symptoms in the presence of negative blood-films were given a series of treatments with Bayer 205 plus 7 grains of tartar emetic. On April 1, 1930, another series of intravenous treatments was administered, using Bayer 205 and tartar emetic. (Three hundred grams of Bayer 205 and 800 grains of tartar emetic were given to 144 animals).
>
> Ten animals were treated again on April 8, 1930, and 111 treated on April 14, 1930, while 106 received another course on April 22, 1930. Due to induration and neck abscesses at the site of former intravenous treatments, 33 animals were not treated on April 14, and 28 were not treated on April 22 for the same reason. Some of these animals died or were killed but the rest were given treatments after the lesions of the neck had subsided. Following the treatment thus described, a few new cases and several relapses developed and general treatment again was given the herd by Dr. C. C. Clay, on October 23, 1930, October 31, 1930, and November 14, 1930, but tartar emetic was used with Bayer 205 only on animals positive for the disease, while Bayer 205 alone was given for prophylactic reasons to the animals with negative blood-films. Now and then an animal or two has had treatments given as late in our period of observation as August, 1931.

It is extremely difficult, even in expert hands, to treat a large herd of animals intravenously without allowing a little of the treatment to escape from the puncture in the vein after the needle has been withdrawn. Any escape of tartar emetic produces very unsatisfactory local results and not infrequently mural thrombi develop in the jugular vein. Sometimes only a patch of slightly indurated subcutaneous tissue occurred and that soon subsided. Many times large abscesses formed with an asso-

ciated jugular thrombosis and occasionally there was a fatal hemorrhage from the vein. This discouraged the curative as well as the prophylactic application of the drug, yet tartar emetic still seems to be the most effective trypanocide.

The greater part of the discouraging results of intravenous treatment with tartar emetic occurred in the young and unmanageable animals. (See table II.)

TABLE II—*Neck injuries following intravenous injections.*

AGE	ANIMALS TREATED	NECK INJURIES	
		NUMBER	%
Under 12 months	25	13	52.0
1 year to 4 years	66	17	25.7
5 years to 20 years	67	6	8.9
Totals	158	36	22.7

Twelve of these cases revealed only a patch of induration or non-discharging abscess. Sixteen animals had discharging abscesses. Seven animals had large open ulcers, jugular thrombosis and severe hemorrhage from the vein. One animal had a most extensive phlegmonous lesion of both sides of the neck and almost complete obstructive thrombosis of both jugular veins.

Recovery is apparently complete in 25 of these animals, but these lesions played an important primary or contributory rôle in the cause of death of eleven animals. Twenty-one of these accidents were in animals only under treatment for prophylactic reasons.

Table III shows that 51 cases were discovered by the first thick-blood-film survey and that in the 32 months which have followed there were 16 new cases and 16 relapses or the recurrence of parasites in the blood-films discovered. At the close of September, 1932, there were 34 cases of treated trypanosomiasis still in the Miraflores herd and apparently they are in good condition except for some that were killed for experimental reasons. Twenty-one (31.3 per cent) of the 67 cases never were found positive for trypanosomes after a full course of treatment had been given.

The cattle that had ranged with this herd were segregated in pastures several miles distant in November, 1931. It is interesting to note that no new cases have developed since then. This

offers supportive evidence that *cattle carriers* were infecting some of the vampire bats of that region.

Relapse occurred in 23.2 per cent of the horses and in 25 per cent of the mules. A gelding, three years old, under prophylactic treatment from January, 1930, to August, 1931, became positive for trypanosomes at the end of that period.

There have been 108 animals added to this original herd of 158 from January, 1930, to September 30, 1932. Sixteen of them

TABLE III—*Trypanosomes in blood-film survey by months (Miraflores Stock Farm herd).*

MONTH	HORSES	MULES	BURROS	TOTAL NEW CASES
1930				
January	44	7	0	51
February	0	0	0	0
March	3 n 5 r	0	0	3
April	4 n 6 r	0 1 r	0	4
May	0	0	0	0
June	0	0	0	0
July	0 1 r	0	0	0
August	0	0 1 r	0	0
September	2 n	0	0	2
October	2 n	1 n	0	3
November	0	0	0	0
December	0	0	0	0
1931				
January	0 1 r	0	0	0
February	0	0	0	0
March	0	0	0	0
April	0	0	0	0
May	0	0	0	0
June	0	0	0	0
July	0	0	0	0
August	1 n	0	0	1
September	2 n 1 r	0	0	2
October	1 n	0	0	1
November	0	0	0	0
December	0	0	0	0
1932				
January	0	0	0	0
February	0	0	0	0
March	0	0	0	0
April	0	0	0	0
May	0	0	0	0
June	0	0	0	0
July	0	0	0	0
August	0	0	0	0
September	0	0	0	0
Totals	59	8	0	67

n = new case.
r = relapse or recurrence of parasites.

were colts that are offsprings of the herd and all were negative and have remained negative to blood-film surveys. There were six colts, eight mule colts and two burro colts. The old animals added were 61 horses and 31 mules. Since September, 1931, an

TABLE IV—*General incidence of trypanosomiasis in Miraflores herd (thick-blood-film survey).*

ANIMALS	NUMBER EXAMINED	POSITIVE	
		NUMBER	%
Horses	138	56	40.5
Mules	16	8	50.0
Burros	4	0	0.0
Totals	158	64	40.5

TABLE V—*Deaths from all causes in Miraflores herd (January, 1930, to August, 1931).*

CAUSE OF DEATH	ANIMALS
Trypanosomiasis, uncomplicated	17
Trypanosomiasis with treatment sequelae or some form of external violence	15
Prophylactic treatment sequelae	8
Other causes unassociated with trypanosomiasis, old animals, unserviceable, etc	17
Total	57

old member of the herd relapsed and one old member became positive for the first time on September 24, 1931. Two of the new horses added to the herd became positive so soon after their arrival that it is difficult to say where they acquired the disease. The others were negative and have not contracted the disease by contact in pastures with the old herd.

DIAGNOSTIC METHODS USED ON MIRAFLORES HERD

We depended on monthly thick-blood-film surveys for the herd in general and on daily films for a week or ten days on clinical suspects. In addition, a guinea pig was inoculated by the intraperitoneal route with 2 to 4 cc of blood, using one pig to each horse. On only one occasion have we had a guinea pig develop the disease from equines in which we failed to find the parasite in the blood-film. At the close of a year of observation, we collected serum from the application of the complement-fixation

test (January 27, 1931) on 123 members of the Miraflores Stock Farm herd. Each specimen was carbolized 0.5 per cent and forwarded in refrigeration to the Bureau of Animal Industry, Washington, D. C. These specimens were divided on their arrival so that Major R. A. Kelser, V. C., U. S. A., Chief, Veterinary Laboratory Section, Army Medical Center, Washington, D. C., also could examine them. A number of these specimens were anticomplementary or otherwise unsatisfactory. Specimens were again collected on March 13, 1931, and sent to the same laboratories. These animals represent the survivors of the herd that had been under treatment for a year. Both laboratories made three separate tests of each serum, using *T. equiperdum* antigens that were about two weeks old. The second shipment of specimens were set up against freshly prepared antigens of *T. hippicum* as well as *T. equiperdum*. These antigens, when of the same age and density, can be expected to give the same results. A comparison of the results obtained from the combined records of monthly blood-film surveys and the complement-fixation test is given in table VI.

It must be stated that since these tests were performed, horse 17 became positive for trypanosomes (August, 1931).

In order to eliminate the confusion caused by the application of the complement-fixation test to a herd that had been under treatment for a long time, we checked these methods on another herd in the early stage of the disease and which had not been given treatment. This Escobal Farm herd (Standard Fruit and Steamship Company) was located on the west bank of Gatun Lake and was negative to a blood-film survey in August, 1930, but by May, 1931, the disease had appeared there. We repeated the blood-film surveys and also collected specimens of serum from 108 animals for the same laboratories to apply the complement-fixation tests. The results are given in table VII.

Note that all but two of this herd were mules. The Bureau of Animal Industry used a one-day-old antigen of *T. equiperdum* and a six-day-old antigen of *T. hippicum*, without getting important variations in the results of the test.

The Army Medical Center used *T. equiperdum* and *T. hippicum* antigens, each three days old, without appreciable variations in the result.

Both laboratories secured a positive diagnosis in mule 93. This animal showed ample clinical evidence of the late stage of the disease but numerous blood-films were examined and found negative for trypanosomes. On the other hand, mule 90 was

TABLE VI—*Comparison of results obtained from the combined records of monthly blood-film surveys and complement-fixation tests.*

LAB. NO.	ANIMAL	TRYPANOSOMES IN THICK BLOOD-FILM	COMPLEMENT-FIXATION TEST	
			B. A. I.	ARMY M. C.
6	Horse	−	−	−
7	Horse	−	−	−
8	Horse	−	−	−
9	Horse	−	−	−
10	Horse	−	−	−
11	Horse	−	−	−
12	Horse	−	−	−
14	Horse	−	−	−
15	Horse	−	−	−
16	Horse	−	−	−
17	Horse	+	−	−
17X	Horse	−	−	−
21	Horse	−	−	−
22	Horse	−	−	−
30	Horse	+	++++	++++
32	Horse	+	−	−
35	Horse	+	−	−
37	Horse	+	−	−
38	Horse	+	−	−
39	Horse	−	−	−
41	Horse	+	−	−
42	Horse	+	−	−
44	Horse	+	−	−
46	Horse	+	−	++++
47	Horse	+	−	−
48	Horse	+	−	−
49	Horse	+	−	−
51	Horse	+	V. S. R.*	+−
52	Horse	+	−	−
53	Horse	+	V. S. R.*	+−
54	Horse	+	−	−
55	Horse	+	−	−
56	Horse	+	−	−
58	Horse	+	−	−
59	Horse	−	−	−
60	Horse	+	−	−
61	Horse	+	−	−
62	Horse	−	−	−
63	Horse	−	−	−
64	Horse	−	−	−
65	Horse	+	−	−
66	Horse	−	−	−
68	Horse	−	−	−
69	Horse	−	−	−
70	Horse	+	−	−
71	Horse	+	−	−
76	Horse	−	−	−
77	Horse	−	−	−
78	Horse	−	−	−
80	Horse	−	−	−
81	Horse	−	−	−
82	Horse	+	++++	++++

*Very slight reaction.

TABLE VI—*Comparison of results obtained from the combined records of monthly blood-film surveys and complement-fixation tests—Continued.*

LAB. NO.	ANIMAL	TRYPANOSOMES IN THICK BLOOD-FILM	COMPLEMENT-FIXATION TEST	
			B. A. I.	ARMY M. C.
83	Horse	−	−	−
84	Horse	−	−	−
85	Horse	+	−	−
86	Horse	−	−	−
87	Horse	−	−	−
88	Horse	+	++++	++++
89	Horse	−	−	−
90	Horse	+	−	−
92	Horse	−	−	−
94	Horse	+	−	−
95	Horse	−	−	−
96	Horse	−	−	−
97	Horse	g. p. +	−	−
98	Horse	−	−	−
99	Horse	−	−	−
101	Horse	−	−	−
102	Horse	−	−	−
103	Horse	−	−	−
106	Horse	−	−	−
109	Horse	−	−	−
110	Horse	−	−	−
112	Horse	−	−	−
113	Horse	−	−	−
121	Horse	−	−	−
123	Horse	−	−	−
124	Horse	−	−	−
125	Horse	−	−	−
126	Horse	−	−	−
127	Horse	−	−	−
129	Horse	−	−	−
131	Horse	−	−	−
132	Horse	−	−	−
133	Horse	−	V. S. R.*	−
134	Horse	−	−	−
136	Horse	−	−	−
138	Horse	−	−	−
139	Horse	−	++++	++++
140	Horse	−	−	−
141	Horse	−	−	−
142	Horse	−	−	−
145	Horse	−	−	−
146	Horse	+	−	−
156	Horse	−	−	++++
160	Horse	−	−	−
161	Horse	−	−	−
162	Horse	−	++++	++++
163	Horse	−	++++	++++
169	Horse	−	−	−
171	Horse	−	−	−
172	Horse	+	−	−
174	Horse	−	−	−
176	Horse	−	−	−

*Very slight reaction.

TABLE VI—*Comparison of results obtained from the combined records of monthly blood-film surveys and complement-fixation tests—Concluded.*

Lab. No.	Animal	Trypanosomes in Thick Blood-Film	Complement-Fixation Test	
			B. A. I.	Army M. C.
177	Horse	−	−	−
178	Horse	−	−	−
25	Mule	+	−	−
72	Burro	−	−	−
73	Burro	−	−	−
74	Burro	−	−	−
114	Mule	+	−	−
116	Mule	−	−	−
118	Mule	−	−	−
122	Mule	+	−	No test
143	Mule	−	−	−
151	Mule	−	−	−
159	Burro	−	−	+−
167	Mule	−	−	−
168	Mule	−	−	−
170	Mule	−	−	−
173	Mule	−	−	−
179	Mule	−	−	−
181	Mule	−	+	−

diagnosed easily by the use of the blood-film and was negative to the complement-fixation tests of both laboratories.

Both the blood-film survey and the complement-fixation test should be used for diagnostic purposes, and in special cases a guinea-pig inoculation should be added. However, it seldom happens that a few frequently repeated thick-blood-film examinations fail to establish a diagnosis in our local form of the disease. It is not an easy nor an inexpensive matter to keep a fresh satisfactory antigen for daily use, yet in the late stage of a chronic or treated case one must have recourse to this diagnostic method. In order to learn how early the complement-fixation test would definitely respond to the disease we inoculated a normal horse and mule with our local strain of *T. hippicum*. On May 25, 1931, these animals were given intravenous injections of guinea-pig blood that contained an abundance of the parasites. The mule was given 2 cc and the horse 2.5 cc of this blood and both animals were positive for trypanosomes the next day. The mule had eight parasites in its thick-blood film and the horse ten parasites.

Subcutaneous inoculations usually require five to seven days before parasites are found in the blood-films. These animals were allowed to run an untreated course for several weeks. Blood

TABLE VII—*Comparison of results obtained from the combined records of monthly blood-film surveys and complement-fixation tests (recheck).*

LAB. NO.	ANIMAL	TRYPANASOMES IN THICK BLOOD-FILM	COMPLEMENT-FIXATION TEST	
			B. A. I.	Army M. C.
1	Mule	–	–	–
2	Mule	–	–	–
3	Mule	–	–	–
4	Mule	–	–	–
5	Mule	+	++++	++++
6	Mule	–	–	–
7	Mule	–	–	–
8	Mule	–	–	–
9	Mule	+	++++	++++
10	Mule	–	–	–
11	Mule	–	–	–
12	Mule	–	–	–
13	Mule	–	–	–
14	Mule	–	–	ac.*
15	Mule	–	–	–
16	Mule	–	–	–
17	Mule	–	–	–
18	Mule	–	–	–
19	Mule	–	–	–
20	Mule	–	–	–
21	Mule	–	–	–
22	Mule	–	+	–
23	Mule	–	–	–
24	Mule	–	–	–
25	Mule	–	–	–
26	Mule	–	++	+
27	Mule	–	–	–
28	Mule	–	–	–
29	Mule	–	–	–
30	Mule	–	–	–
31	Mule	–	–	–
32	Mule	–	–	–
33	Mule	–	–	–
34	Mule	–	–	–
35	Mule	–	–	–
36	Mule	+	++++	++++
37	Mule	–	–	ac.*
38	Mule	–	–	–
39	Mule	–	–	–
40	Mule	–	–	–
41	Mule	–	–	–
42	Mule	–	–	–
43	Mule	–	–	–
44	Mule	–	–	–
45	Mule	–	–	–
46	Mule	–	–	–
47	Mule	–	–	–
48	Mule	–	–	–
49	Mule	–	–	–
50	Mule	–	–	–
51	Mule	–	–	–
52	Mule	–	–	–
53	Horse (gelding)	–	–	–
54	Mule	–	+	++

*Anticomplementary.

TABLE VII—*Comparison of results obtained from the combined records of monthly blood-film surveys and complement-fixation tests (recheck)—Concluded.*

Lab. No.	Animal	Trypanasomes in Thick Blood-Film	Complement-Fixation Test	
			B. A. I.	Army M. C.
55	Mule	–	–	–
56	Mule	–	–	–
57	Mule	–	–	–
58	Mule	–	–	–
59	Mule	–	–	–
60	Mule	–	–	–
61	Mule	–	–	–
62	Mule	–	–	–
63	Mule	–	–	–
64	Mule	–	–	–
65	Mule	–	–	–
66	Mule	–	No record	–
67	Mule	–	–	–
68	Mule	–	–	++++
69	Mule	–	–	–
70	Mule	–	–	–
71	Mule	–	No test	–
72	Mule	–	–	–
73	Mule	–	–	–
74	Mule	–	–	–
75	Mule	–	–	–
76	Mule	–	–	–
77	Mule	–	–	–
78	Mule	–	–	ac.*
79	Mule	–	–	–
80	Mule	–	–	–
81	Mule	–	–	–
82	Mule	–	–	–
83	Mule	–	–	–
84	Mule	–	–	–
85	Mule	–	–	–
86	Mule	–	–	–
87	Mule	+	++++	++++
88	Mule	+	++++	++++
89	Mule	+	++++	++++
90	Mule	+	–	–
91	Mule	+	++++	++++
92	Mule	+	++++	++++
93	Mule	–	++++	++++
94	Mule	+	No test	+++
95	Mule	+	++++	++++
96	Mule	+	++++	++++
97	Mule	+	++++	++++
98	Mule	–	+	–
99	Mule	+	++++	++++
100	Mule	–	–	–
101	Horse (gelding)	–	++++ (Control sl.ac.*)	++++
102	Mule	–	–	–
103	Mule	+	++++	++++
104	Mule	+	No specimen	No specimen
105	Mule	+	No specimen	No specimen
106	Mule	–	No specimen	No specimen
107	Mule	–	No specimen	No specimen
108	Mule	–	No specimen	No specimen

*Anticomplementary.

specimens were collected for the application of the complement-fixation test, 16, 24, 31 and 38 days after the two animals had been inoculated. These were sent to the same laboratories for examination and each specimen was reported as positive for the disease. We had not anticipated so early a response to the test or earlier specimens would have been forwarded. Both animals revealed a very definite clinical picture by the time the third specimen was forwarded. The horse, alone, showed marked edema of the belly. These animals were now placed on treatment using tryparsamide. Five human doses (10 grams) were taken as one dose for an animal. This treatment has been continued every five to seven days during a period of six weeks. Their physical condition has improved but daily blood-films have been positive in almost every instance. The parasites are present in a sparse manner as a rule, but about once a week they are found in great numbers.

On August 13, 1931, the mule had over 200 parasites in one thick film while the horse, on August 17, 1931, had over 800 parasites in its film. Treatment was started on the horse on June 29, 1931, and on the mule on July 6, 1931. The dose of the drug was increased to 12 grams. The daily blood-film continued to be positive for trypanosomes up to September 22, 1931, when the treatment was changed to a series of three doses of Bayer 205 plus tartar emetic. The mule showed a few trypanosomes for the next two days after the first dose, but both the horse and mule remained steadily negative from that date forward. The mule, at the time this is written (October, 1932), is in good physical condition and is performing the duty of a saddle animal for the Cattle Industry. The horse later was used in an experiment to see whether the disease and its treatment developed any degree of immunity against a new infection by the same parasite. It was given 2 cc of blood from the heart of guinea pig 51 that was positive for *T. hippicum*. This was an intrajugular injection, made January 24, 1932. It again acquired the infection but never revealed many trypanosomes in its blood-films. The clinical picture of the disease reappeared and its condition became so helpless that on March 11, 1932, we killed it.

Associated Diseases Found

The general application of the thick-blood film during the first 19 months of observation of the Miraflores herd shows that 27.8 per cent had piroplasmosis and 20.2 per cent had filariasis. It is safe to conclude that every animal raised on the ranges in this

region becomes a carrier of these diseases as soon as it becomes a few months old. The only acute cases of piroplasmosis that we find are those which occur in recently imported horses used as mounts for the Army and in young colts and foals in the rural herds. Almost every autopsy examination reveals several adult filaria in the peritoneal cavity.

Spirochetosis: The general surveys of the herds in the Republic of Panama and the Canal Zone revealed four animals positive for this disease. Three of them were very scant infections, while one was very abundant. We do not believe that the infection is very important from the viewpoint of the clinician.

Laminitis: This was found in its usual incidence among old animals and the lesion seems to be provoked or intensified by the use of trypanocidal drugs. Indeed, some cases of laminitis appear to be directly caused by the drug. These drugs also caused many animals to be stiff and lame for a day or two after treatment. Many animals also developed a dry painful swelling of the anal and vulvar tissues, with radial fissures that oozed and required attention to prevent myiasis. Two animals did develop anal myiasis (*Cochliomyia macellaria* Fab.). Two abortions occurred following treatment but the disease may have been as much responsible for these accidents as the drug.

Intestinal parasites: These are, of course, very common and many extremely heavy infestations were found in animals from six months to two years of age. Strongylidosis, as a severe infection, has been present in every horse and mule over a few months of age that the senior author has examined at autopsy during the past three years in various parts of Panama and the Canal Zone. Every autopsy has revealed either a verminous aneurysm of the mesenteric artery or chronic obliterative arteritis of this vessel with marked stenosis. We are inclined to attach more importance to this arterial lesion than is usually given it by writers. One must admit at once that a great many animals manage to lead a long life with perhaps few serious periods of interference from this disease. On the other hand, many serious complications do occur and these are probably more common in young adult animals. Serious accidents are just as apt to be associated with small aneurysms as large ones. These lesions of the mesenteric artery are very easy to overlook at autopsy because they usually form a mass about the size and shape of a lemon that is buried in the mesenteric tissues and surrounded by a cluster of enlarged lymph-nodes. The aneurysms

are located usually at a distance of a few centimeters from the wall of the aorta.

We do not feel that "colic" is the most frequent clinical manifestation of the disorder. Unexplained emaciation, anemia and weakness in young animals seem to be the best guide to a clinical diagnosis. A vast number of these aneurysmal sacs, in animals acutely ill as a result of this disease, are filled with either organized or recently formed thrombi as well as the larvae of the parasites. The sac, when emptied, very frequently shows one or more necrotic spots in some part of its wall that involves all coats of the vessel. These necrotic areas are usually well supported against rupture by the acute and chronic inflammatory processes set up in the tissues immediately outside the sac. This disease of the mesenteric artery is a great handicap to a herd when it is invaded by other diseases and particularly by trypanosomiasis. The latter disease is accompanied by severe anemia as well as by the increased danger from thrombosis due to the toxic effects of trypanocidal drugs.

We have seen no evidence at autopsy that the three trypanocidal drugs we employed were effective against the larvae in the aneurysmal sacs. We feel reasonably certain that nine deaths in the Miraflores herd can be ascribed chiefly to accidents in verminous aneurysms of the mesenteric artery, such as embolism and thrombosis and some obstruction to the arterial blood supply which must in turn have interfered with the assimilation of food. An effective treatment directed at the larval stage of these worms might also enhance the danger of embolism since the dead larvae may become foreign bodies in the blood-stream. We have accepted a number of opportunities to examine animals killed because they were unserviceable, that is, no definite reason could be assigned for their condition, such as the presence of a well-defined disease or injury or advanced age. All of these animals revealed either stenosis or dilatation of the mesenteric artery of a severe degree due to strongylidosis. There was marked interference with the function of this arterial tree.

Many young animals in the Miraflores herd were treated for worms by the oral administration of tartar emetic and in addition they received routine treatment intravenously with Bayer 205 plus tartar emetic for trypanosomiasis. We sacrificed some of these animals to note the result of treatment and were disappointed when we found living larvae in the aneurysmal sacs and also many living parasites fast to the mucosa of the cecum. It is also worthy of note that living adult filarial parasites were

found in the peritoneal cavity and that in some instances piroplasms also could be detected in blood-films following treatment.

Prophylactic Treatment

The many evil results following such treatment in the Miraflores herd and the need of very frequent treatments actually to prevent infection have caused us to decide that such management of a herd in an epizoötic is not warranted. Furthermore, the expense involved would soon be out of all proportion to the market value of the animals.

Segregation

A diligent laboratory service soon can weed out the positive animals so that segregation and treatment can remove the risk of the spread of the disease from such animals. Protection of the negative animals can be secured by stabling them in illuminated or screened barns. The infected bats will live only about a month and the only "carrier risks" left at large are the cattle and other animals already pointed out that become light carriers of the parasites for a few months. We have known for many years that animals kept continuously in stables seldom develop trypanosomiasis. They are far more apt to be free from the attacks of bats, horse-flies and other insects, as well as exposed to a much less extent to the mechanical means of transmission. Even pasture segregation in a wide agricultural region can be of considerable benefit. The Miraflores herd was found divided in four pastures relatively close together around the same arm of Miraflores Lake. All of these pastures developed some cases of trypanosomiasis, but a very large majority of the cases came from one pasture. Segregation is an easy matter with local Army animals for they are kept in stables lighted by electricity and are never on pasture. Even during maneuvers through the jungle and the interior these animals are kept on a picket line and fed, and to some extent protected by lanterns. They avoid as far as possible any contact with range or village animals. Animals kept in this manner are open to daily inspection and a sick horse or mule is found in an early stage of its illness and placed in a quarantined stall. These animals are well groomed, scores are under daily treatment and ticks removed and their bites treated. There are three collections of Army animals on the east bank of the Panama Canal directly opposite two foci of the disease on the west bank of the canal. One period of Army maneuvers has occurred during the epizoötic. Notwith-

standing these conditions, we have had but five cases of the disease in almost 2,000 Army animals.

Strict quarantine of the diseased areas, of course, was of some assistance. We were not so sure what results might be expected in rural animals that were kept part of the time in stables and part of the time in pastures. An owner is in great difficulty when this disease is present in a herd, since he cannot safely add replacement animals during an epizoötic and he dislikes sacrificing animals that the microscopic examination alone shows are positive and in which there is no sign of illness to be found on inspection or by the use of the animal. He receives no pay for the animals killed under these circumstances.

The Escobal Farm of the Standard Fruit and Steamship Company had a herd of this character located on the west bank of Gatun Lake about 45 minutes launch ride from Gatun, the nearest Canal Zone village. We performed ten thick-blood-film surveys at intervals of about three days and applied one complement-fixation test to the herd. The positive cases were collected as they were discovered in one small banana farm about four miles from the clean members of the herd. There was a ridge of about 400 feet elevation between the clean and infected animals. All late-stage cases of the disease were killed, but the other positives were worked until they became unserviceable before they were sacrificed. Some of these animals were of service for two or three months. Three of them, small mules, are still living at the end of 16 months, but are in very poor condition. No new cases have appeared in the clean members of the herd nor in the new animals added to the main herd. It is of interest to note that almost no cattle ranged the area in which this herd was used.

A second herd located not far away contained a smaller collection of animals. We have adopted the same method of management except that all positive cases were killed and this area also has remained free of the disease. It would appear from these experiences that mechanical means of transmission should be given due consideration as well as the natural vector. The perpetuation of the disease in a region is no doubt due to such a vector and some isolated animal reservoirs. The senior author has placed a guinea pig with clipped ears in a closed pen with one infected guinea pig with a clipped bleeding ear and managed to infect the clean pig. This cannot happen with much success unless straw or grass is placed on top of them in crowded quarters. This forces them to run in tunnels through such grass

or straw and they pick up wet fresh blood on the freshly clipped ear.

During the construction period of the Panama Canal we were inclined to believe that this disease would be eradicated in the Canal Zone within a few years. We based this belief on the hope that the Canal Zone would remain a depopulated region for domestic animals as well as man and that transportation would be motorized to such a large extent that the disease could not perpetuate itself in the small number of scattered stabled animals required by the Panama Canal and the United States Army. The period of operation and maintenance of the Panama Canal has now been in effect many years and equine trypanosomiasis is still present and reaches at times epizoötic proportions.

The idea of a depopulated Canal Zone was abandoned. Farms, large and small, are scattered all over the Canal Zone and herds of animals are found in villages along the boundary. Trial systems connect certain parts of the Canal Zone with these contact villages. It is more or less easy to control the disease in animals under the direction of organizations like the Army and the Panama Canal and large agricultural organizations but it is difficult to follow the condition of each small private owner's stock. Such animals are potential reservoirs that can remain concealed for a long time. The Panama Canal, the Army and large agricultural organizations have learned from experience that they cannot motorize to the extent they planned a few years ago. It is certain that a relatively large number of horses and mules will always be required. The difficulty in maintaining control of the disease is greater at present than in the past because of the road-building which permits easy and inexpensive transportation of cheap work animals, a class that formerly was not worth ocean transportation.

Trypanosomiasis has seldom gained a foothold on the west side of the Canal Zone. The new road system now open for use through the upper provinces of Panama already has permitted the disease to become established about 200 miles beyond the west side of the Canal Zone. These roads soon will form a segment of the international highway and when this is in operation there is no reason why this form of trypanosomiasis should not find its way by relay through Central America to the United States unless vigorous steps are taken to check its advance. The senior author has had opportunities to survey large herds of stock in the Atlantic coastal plains of Costa Rica, Honduras and Guatemala, but did not find a case of the disease in these regions. It was found on the Atlantic coast of Colombia.

A closely related equine trypanosomiasis, dourine,[5] in the past has appeared in nine of the middle west and southwestern states as well as Canada and the time and expense involved in its control should focus serious attention on the possibility of this form being introduced.

There are many animals, particularly mules, afflicted with an early stage of the disease that could travel the road system from Panama to Texas before the disease would plainly manifest itself to a casual examination by an inspector. Diagnosis depends chiefly on laboratory methods in the early stage of the disease.

SUMMARY

1. The first scientific record made in regard to the presence of equine trypanosomiasis (murrina or derrengadera) on the isthmus of Panama was written by Darling, in 1910. He established the fact that it was a trypanosomiasis and he named the pathogenic agent *Trypanosoma hippicum*. The clinical character of the disease and losses due to it seem to have been known long before this date. The local belief is that epizoötics of this character did not occur in Panama until large business contacts with other nations started the importation of stock. Whatever the facts may be regarding introduction from outside, it is certain that the disease has been enzoötic for a very long period of time in that part of the republic which includes the Canal Zone and extends to the boundary of Colombia. Outbreaks of the disease have not appeared in Panama during the last 22 years with seasonal regularity, yet local stock-owners state that most of the widespread epizoötics have made their appearance in the months from September to January. There is usually a period of a few years between epizoötics. We believe this is due in part to the slow replacement of animals killed by an epizoötic, thus greatly reducing the number of contacts and exposures. New cases and epizoötics of major or minor degree, in our experience, have occurred in every month of the year. The disease appeared again in epizoötic manner in the Panama Canal Zone during the close of the year 1929. There were two foci on the west bank of the Canal Zone. One occurred at the Mariflores Stock Farm, near the Pacific terminus of the canal, while the other appeared at the Atlantic terminus, between the Gatun Dam and Cristobal. A focus also was found about 200 miles west of the Canal Zone in the republic of Panama. During the late months of 1931 and the early months of 1932, it ap-

peared about 30 miles down the Pacific coast from the city of Panama, in the Sabanas between Pacora and Chepo.

2. *Trypanosoma hippicum* and the disease it produces seems to us to correspond to that found in Colombia. Now that we have learned that *T. hippicum* can be carried by cattle, there is more supportive evidence that inclines us to share Wenyon's believe that *T. hippicum* and *T. venezuelense* are simply races of *T. evansi* and that they differ from it only in minor details.

3. The clinical picture of the disease is exhibited by fever of an irregular character, progressive emaciation, anemia, faint icterus, a rough coat and sometimes edema of the most dependent part of the abdominal wall and sheath and to a less extent of the legs. Late in the illness there is a marked weakness of the posterior extremities and the animal walks with a stiff, staggering gait, frequently dragging the foot. There is no impairment of appetite throughout the course of the disease. It is frequently possible to find the trypanosome in the blood-film of an animal two or three weeks before clinical evidence of the disease appears. For this season, as soon as a single case is recognized, a general laboratory survey of the herd should be made. Such a survey may consist of blood-film examinations for the detection of trypanosomes, inoculations of susceptible animals and the application of the complement-fixation test for equine trypanosomiasis. We select the thick-blood-film method for rapid field surveys, since it is difficult to examine fresh blood-films in the field in cloudy, rainy, windy weather. A herd should be examined every two or three days, for at least ten surveys, in order to pick out all of the cases that are active or are in the incubation period. When it is possible to have recourse to the complement-fixation test, this will be of great assistance but in most field work where we have daily access to a herd we prefer the blood-film survey, supported by guinea-pig inoculations in selected cases.

4. The recent work of Dunn[6] in his study of the local bats, has established beyond doubt that the vampire bat, *Desmodus rotundus murinus* Wagner, is an important natural vector of this disease and that it can disseminate the disease on its nightly feedings over an average period of about one month after it has acquired the disease and before death overtakes it as a result of the disease. It adds additional proof that this trypanosome can pass through the normal mucosa of the alimentary tract. The question of natural insect vectors (horse-flies) is still open. However, our epizoötics of this disease are found only in herds

where the disease has been given several months to spread and in the case of an insect vector the epizoötic should appear in an explosive manner. We believe that the vampire bat is probably the main vector that keeps the disease enzoötic. Mechanical means for the spread of the disease must be given due consideration, for we know that any means of transferring wet, infected blood to breaks in the skin of a negative animal or into the mucous tracts of an animal can convey the disease. Coition is probably as important a means of transfer as any other mechanical means.

5. We can experimentally produce this form of trypanosomiasis in all of the wild and domestic animals that we have been able to collect, with the exception of the chicken. Nearly all of them, however, die within a few weeks after they acquire the infection. The small, native mule, domestic cat, white-tailed deer, brocket deer and collared peccary (wild hog) can live from four to 16 months before death supervenes. In fact, we have three untreated mules that have survived 16 months and, although their physical condition is very poor, they will probably live several months longer. Animals that we have been able to infect but which recover spontaneously are the goat, sheep, domestic hog and a calf. *The only animals, aside from the equines, that we have found carrying a naturally acquired infection, were cattle.* Such stock ranging with an infected herd of horses and mules, when tested by guinea-pig inoculations, revealed the fact that 4.5 per cent *carried* this form of trypanosomiasis. Where equines and cattle range together day and night, without protection from vampire bats, the question of control of this form of trypanosomiasis becomes a serious problem to the owner. We think it safe to assume that *animal reservoirs* or *carriers* of great importance for the perpetuation of the disease in Panama are cattle and certain horses, mules and burros with a high individual resistance. Reservoirs of secondary importance are the wild and domestic hogs, deer, sheep, goat and the domestic cat.

6. Immunity is not conferred by infected guinea pigs to their offspring and the foals of infected mares can acquire the disease. One experiment horse, that was not treated until it was in a critical advanced stage of the disease, apparently was cured. Its physical condition was good and guinea pigs inoculated with blood from its jugular vein did not acquire the disease. We successfully reinfected this animal and it died of the disease,

although it never had the abundant number of parasites that had appeared in the initial infection.

7. The strains of *T. hippicum* do not always behave in the same manner on initial infections secured in guinea pigs from different horses and mules, but this may be due to individual peculiarities in the animals, since, when carried through a long series of guinea pigs, the average duration of the disease is about the same. The strain secured from *cattle carriers* of the disease ran a typical clinical course of the disease, ending in death in the average time expected. The trypanosome was similar in all respects to *T. hippicum* recovered from horses and mules in an epizoötic except that the majority of the parasites revealed either a very small centrosome or none at all. The introduction of cattle, horses and mules from all parts of the world and, in particular, from the north coast of South America should have succeeded in bringing in almost any form of trypanosomiasis that could survive the period of transportation by sea. This is another reason for considering the possibility of a very close relationship between all our tropical and subtropical forms of equine trypanosomiasis common to the western hemisphere.

8. No treatment was in local use for the disease until a few years ago. It was simply a question of surveying all animals and then quarantining or killing the positives found. A number of new trypanocidal drugs have appeared in the past fifteen years and the Miraflores epizoötic gave us a chance to try some of these. We were forced to choose those that happened to be in stock on the Isthmus and these were tryparsamide, naganol (Bayer 205 vet.) and tartar emetic. Tryparsamide did not kill the infection even over a long course of treatment in a horse and mule, but it seemed to offer some protection, since the animals ran a longer course than usual without getting off their feet. The use of naganol gave better results but was followed by too many relapses. Our past experience with tartar emetic proved to us that it was an effective trypanocide but the drug was too toxic for the host. We compromised by using a half-dose of tartar emetic with a full dose of naganol. There seems to be something in naganol that guards the action of tartar emetic. At any rate it is the first time that we have been able to treat any animal with the disease successfully. It is our practice at present to treat them as follows: Horses and mules over 600 lbs. in weight receive 8 grams of naganol and 21 grains of tartar emetic divided into three doses. The

first dose consists of 4 grams of naganol and 7 grains of tartar emetic; the second dose is 2 grams of naganol and 7 grains of tartar emetic and the third dose is the same as the second. Each dose is dissolved in 200 cc of distilled water and injected into the jugular vein by gravity. Six days intervene between these injections or doses. It is extremely difficult, even in expert hands, to treat a large herd of animals in this manner without the escape of some of the drugs from the punctured vein. Bad results follow the least escape of the drug. Sometimes only a patch of slightly indurated tissue occurred that quickly subsided. Several times large abscesses formed with an associated jugular thrombosis and occasionally a fatal hemorrhage from the jugular vein. Most of these bad results occurred in young, unmanageable animals. We have ceased using or advising the use of prophylactic treatment since it must be given too frequently, it is too expensive, it is followed by too many bad results and, furthermore, the control of the disease can be accomplished better by other means. The records show 51 cases in 158 animals belonging to the Miraflores herd, when control of the situation was undertaken. In the 32 months of observation that have followed (this includes 108 additions to the herd), 16 new cases have developed and 16 relapses have occurred. At the close of September, 1932, there were 34 of the positive cases looked upon as cured. Some of these were sacrificed for study, while many of them are on duty and seem to be in normal condition. Twenty-one of the positive cases were never found positive after the first full course of three treatments was given. No new cases have developed in the past eleven months. During this period of time no cattle have ranged with the herd. The original herd has received 108 additions since the first diagnostic surveys were made (from February, 1930, to September, 1932). Sixteen of them were colts that are the offspring of the herd, 61 horses and 31 mules. Since September, 1931, an old member of the herd relapsed, one old member became positive for the first time and two additions to the herd became positive so soon after their arrival that doubt exists as to whether they came infected or acquired infection later. Notwithstanding the wonderful advance made in the manufacture of trypanocidal drugs, it is still necessary to seek for something that can be more cheaply, easily and safely administered. The majority of the animals in this region are not valuable enough to warrant the expense of the prophylactic and curative courses as recommended by the manufacturers of these drugs. A safe drug that

can be purchased easily and administered by the average owner of cheap work animals is needed.

9. The general use of the blood-film surveys during the first 19 months of observation of the Miraflores herd showed that 27.8 per cent had chronic piroplasmosis and 20.2 per cent had filariasis. It is safe to conclude that every horse and mule that grows up on the local ranges acquire these infections. The only acute cases of piroplasmosis that we have found were those that occurred in animals recently imported from the United States or colts born on the local farms. Spirochetosis was found four times in our general surveys. We do not consider it very important from the clinical viewpoint. Intestinal parasites are, of course, very common and many heavy infections were found. Strongylidosis, as a severe condition, has been present in every animal above a few months of age. The verminous aneurysms of the mesenteric artery and mesenteric arteritis connected with this infection are serious factors. Many animals lead a long life with this condition present but it is a serious potential source of trouble. At any time the animal is brought below par, a mural thrombosis is apt to develop that partially or completely cuts off the lumen of this important vessel. It is of interest to note that the use of tartar emetic by mouth and by intrajugular injections have in most cases failed to kill larvae in these aneurysms as well as many that attached to the mucosa of the cecum. Osteomalacia (bighead) was recorded in five animals as well developed cases of this disease. Laminitis was found in its usual incidence in old animals. The lesion is intensified by the use of the trypanocidal drugs. Indeed, some cases of acute laminitis appear to be the direct result of the use of the drug.

10. Cardinal features in the management of a herd infected with this disease should consist of numerous laboratory surveys, frequently repeated, employing the blood-film, animal inoculations and the application of the complement-fixation test in order to segregate the positives for treatment and to destroy old or useless animals. Protect mules and horses in screened or illuminated stables from dusk to daylight to prevent bat biting. Avoid ranging horses and mules with cattle. Treat all open sores and cuts to avoid mechanical transfers of the disease from animal to animal and prevent coition until the herd is again clean.

11. Murrina or derrengadera presents a different problem in control measures than dourine. The latter is said to be transmitted almost solely by coition and measures for the suppression

of dourine are directed only at this method of the spread of the disease. Murrina also can be transmitted by coition, but mechanical means, the animal reservoirs and the vampire bat must be given attention in control measures. The disease is not limited to brood mares and stallions. Colts, mules and young geldings become victims of the disease and even cattle are found with natural infections.

12. We have never been able to establish a laboratory diagnosis of this disease west of the Canal Zone until January, 1930, when we found it as far up the Pacific coast as Aguadulce, a distance of about 200 miles. We consider the disease enzoötic in the Canal Zone and that part of the republic of Panama next to Colombia. The completion of the international highway will favor the introduction of this disease northward and conceivably as far north as the United States unless quarantine methods are placed in effect. The disease can become established in a herd several weeks and sometimes months before ordinary inspection of the herd will show a serious loss of animals and sufficiently advanced stages of the disorder to cause a suspicion of its real nature.

ACKNOWLEDGMENTS

The Gorgas Memorial Laboratory wishes to express its thanks to the Health Department and Supply Department of the Panama Canal, to the Panama Canal Department of the United States Army and to the Republic of Panama for the opportunity offered to study this disease. The Supply Department of the Panama Canal, through J. H. K. Humphrey, Assistant Chief Quartermaster, has given us excellent opportunities for a long period of observation of the herd at the Miraflores Stock Farm and he is continuing with us a critical study of the disease. Thanks are due also Dr. C. C. Clay for his assistance in continuing treatment during the absence of Dr. T. L. Casserly. We are deeply indebted also to the Pathological Division of the Bureau of Animal Industry at Washington, D. C., and to the Veterinary Laboratory Section of the Army Medical School, Army Medical Center at Washington, D. C., for their kindness in building fresh antigens and setting up the complement-fixation tests. Our thanks are due the United Fruit Company and Standard Fruit and Steamship Company for their coöperation in the study of their herds.

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CONVENTION NOTES

A quartet of veterinarians registered from each of five states: Alabama, New Jersey, Oklahoma, South Carolina and Wyoming. From *Alabama* came Drs. H. C. Ayer, Decatur; C. A. Cary, H. Odom, Auburn; C. Stewart, Cullman. From *New Jersey* came Drs. Edward L. Baldwin, Newark; E. R. Cushing, Plainfield; J. B. Hagenbuch, Plainsboro; M. L. Morris, New Brunswick. From *Oklahoma* there were Drs. Willis F. Hall, Anadarko; Frank R. Knotts and H. W. Orr, Stillwater; Chester E. Williams, Butler. *South Carolina* sent Drs. W. A. Barnette, Greenwood; M. R. Blackstock, Spartanburg; F. P. Caughman, Columbia; L. J. Hogan, Charleston. The Wyoming quartet consisted of Drs. H. D. Port and W. A. Sullivan, Cheyenne; L. H. Scrivner, Laramie; Major H. K. Moore, Fort Francis E. Warren.

Michigan contributed thirty-seven to the total registration. They were: Drs. J. L. Ackerson, Milan; C. M. Barnum, Kent City; Eldon C. Barclay, Almont; T. L. Bott, Coldwater; F. P. Calkins and E. E. Hamann, Lansing; S. G. Colby, Monroe; H. P. Conrad, Abbottsford; C. C. Dauber, Sturgis; J. LaVere Davidson, Kalamazoo; Glen LeRoy Dunlap, Ann Arbor; S. R. Elkow, Highland Park; A. Elgas, Hartford; Herbert Elzinga, Marne; Ward Giltner, E. T. Hallman, B. J. Killham, W. T. Oglesby, John W. Patton, H. J. Stafseth and Walter W. Thompson, East Lansing; G. H. Gerlach, Morenci; F. K. Hansen, Marquette; William Hansen, Greenville; W. A. Higgins, E. E. Patterson, A. S. Schlingman and L. H. LaFond, Detroit; E. L. Krieger, Benton Harbor; A. Z. Nichols and J. C. Whitney, Hillsdale; Edwin R. Perrin, Northville; R. D. Rice, Maple Rapids; Earl D. Smith, Plainwell; F. E. Stiles, Battle Creek; O. H. Van Brussel, Wayland; L. A. Winter, Eau Claire.

STUDIES ON VACCINES IN INFECTIOUS MASTITIS*

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Mastitis is fast becoming, if it is not already, the most important disease problem with which the dairyman has to deal. In a previous paper[1] the writer has pointed out that mastitis may be of either the infectious or the non-infectious type. The non-infectious mastitis usually yields to local applications and the feeding of the correct amount and proper balance of concentrates. It is the infectious mastitis, caused by streptococci, which is so destructive to the udder tissue. This paper reports a study of vaccines in treating and preventing this type of mastitis.

In the literature are found a few conflicting reports on the use of vaccines in infectious or streptococcic mastitis. Torrey,[2-4] my immediate predecessor in this work, in his annual reports, makes mention of favorable results obtained from vaccination of infected animals. Rosell[5] reported favorable results on the use of a vaccine.

The tools to aid in diagnosis of mastitis as reported by the different workers are numerous and of many types. Each has its value as an aid in diagnosis, but comparison of the results of several tests seems to be necessary to reach a definite conclusion. The writer uses a combination of the periodic bacteriological examination of the milk and physical condition of the udder and milk in diagnosing mastitis. These were used to detect infected animals before vaccination and also to check upon the condition during and after the period of vaccination. The herds used in this study were all privately owned; the owners had noted the physical change in the milk and udders of some of their animals, and came to the laboratory for help. In this manner a varied group of animals was obtained, thus presenting conditions which simulate those found by the practitioner who deals with dairy animals.

Upon the repeated bacteriological examination of the milk and the physical examination of the milk and udder, the prevalence of the disease was determined and made known to the owner. The herd then was placed at the disposal of the writer for a study of vaccine treatment. Repeated bacteriological examinations consisted of culturing milk from each quarter at two or three con-

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secutive weekly intervals. This means of examination proved very accurate in detecting the quarters harboring and eliminating the streptococci capable of producing mastitis. The extent of the disease in the herd, as the owner could detect it, was measured solely by the change in the physical appearance of the milk. Its rapid spread was also quite evident according to the history of the herd as given by the owner and according to the isolation of streptococci when the milk was cultured previous to vaccination. The technic used in collecting and culturing was presented in a previous paper.[6] Weekly bacteriological examination of the milk secretion of each quarter and physical examination of the udder are very useful in following the spread of infection in the herd. The owners were very much interested in this condition and, therefore, kept a record of the daily variations, if any, in the milk and the condition of their cows. In many cases, in the control work previous to vaccination, streptococci were isolated from the quarters of some cows without presenting clinical manifestations. Frequently this milk did not react to the other tests commonly used in detecting mastitis, but in a short time the disease made its unmistakable clinical appearance.

When infectious mastitis was present in some animals of a herd, the owners would insist on treating all the infected animals. This afforded a study of vaccines as curative treatment, but without any infected animals as controls. The negative animals also were vaccinated; this afforded a means of studying the preventive or prophylactic value of vaccination in mastitis. The results of the use of two types of vaccine are reported in this paper.

Herd-Autogenous Vaccine

The first type of vaccine studied was of the herd-autogenous type. The causative organisms were isolated and stock cultures maintained on blood-agar slants. In the preparation of the vaccine, subinoculations were made from the stock cultures to dextrose (1 per cent), veal-infusion broth (pH 7.4) and incubated at 37°C. for 24 hours. The cells in each culture then were concentrated by centrifuging; just sufficient salt solution (0.85 per cent) was added to suspend the sedimented cells in each container. The cells of the different cultures then were transferred aseptically into a vaccine bottle and diluted with sterile physiological (0.85 per cent) salt solution to a reading of one on a Gates nephelometer. At the beginning of the study of this type of vaccine, the cells, for the initial injection, were heat-killed (60°C. for 20 minutes). For the following weekly injec-

tions the cells were grown as before, suspended in physiological salt solution and then phenolated by the addition of 0.5 per cent phenol. In more recent studies the 24-hour-old cells were sedimented by centrifugation, resuspended in a concentrated form in sterile physiological salt solution, and used as a living-culture vaccine. In all cases the vaccines were freshly prepared and used within several hours of the time of preparation.

Experimental Results

At least two, and frequently many more, consecutive weekly examinations were made of the milk and udders before vaccination was begun. These served to indicate the condition in the herd at the time that vaccination was started and they are labeled in tables and graphs as "*beginning.*" Each succeeding week, there was made an additional injection of vaccine in the amount indicated. These were labeled according to the number of weeks after the first injection and examination. Thus in the tables and graphs, "third week" means at the time of the fourth injection and three weeks after the first injection or two weeks after the second injection.

In the initial experiments the vaccine used was always of the heat-killed type. This was changed later, so that cows received one injection of the killed (either by heat or phenol) culture and subsequent injections of the living bacteria. As the work progressed, it was found unnecessary to begin with a killed culture; thus all later studies have been carried out by beginning with a somewhat more dilute living-culture vaccine. During the time of injection of the living culture by the subcutaneous route the animals were watched closely for both local and systemic reactions. No ill effects whatsoever were noted in the animals. For convenience and ease of injection, subcutaneous inoculations were made just posterior to the point of the withers. Special study was made on the effect of the vaccine on the mammary gland itself, but no localized infections in the mammary gland resulted.

In the use of the herd autogenous vaccine, it is of interest and perhaps of importance that each organism used was cultivated separately and, at the time of vaccine preparation, all were placed in the same bottle. This insured sufficient growth of each culture, whereas, if all had been inoculated into one flask, there would have been the possibility that the more vigorously growing streptococci would have crowded out some of the less vigorously growing ones, which also are essential in the vaccine.

TABLE I—*Data on vaccine treatment in herd A.**

TIME OF TREATMENT	VACCINE USED	VACCINE USED (CC)	COWS INFECTED		QUARTERS INFECTED	
			No.	%	No.	%
Beginning..	Phenolized—heat killed	5	3	50.0	4	16.7
1st week....	Phenolized	5	3	50.0	4	16.7
2nd week...	Phenolized	5	3	50.0	3	12.5
3rd week...	Phenolized	5	1	16.7	1	4.2
4th week...	Phenolized	5	1	16.7	1	4.2

*There are six milking cows in this herd. The State Department of Health and the Bureau of Dairying forbade the sale of milk from this herd, but at the end of the fourth week they again allowed the sale of this milk in all cases except the one remaining infected cow.

TABLE II—*Data on vaccine treatment in herd B.**

TIME OF TREATMENT	VACCINE USED	VACCINE USED (CC)	COWS INFECTED		QUARTERS INFECTED	
			No.	%	No.	%
Beginning..	Phenolized—heat killed.	5	6	54.5	13	29.8
1st week....	Phenolized	10	6	54.5	5	11.4
2nd week...	Phenolized	10	4	36.4	5	11.4
3rd week...	Phenolized	8	4	36.4	5	11.4
4th week...	Phenolized	6	4	36.4	5	11.4
5th week...	Phenolized	5	4	36.4	4	9.1
6th week...	Phenolized	5	4	36.4	4	9.1

*There are 11 milking cows in this herd.

TABLE III—*Data on vaccine treatment in herd C.**

TIME OF TREATMENT	VACCINE USED	VACCINE USED (CC)	COWS INFECTED		QUARTERS INFECTED	
			No.	%	No.	%
Beginning..	Phenolized—heat killed.	5	12	30	25	15.6
1st week....	Living, autogenous	7	12	30	19	11.9
2nd week...	Living, autogenous	7	6	15	6	3.8
3rd week...	Living, autogenous	8	4	10	7	4.4
4th week...	Living, autogenous	8	4	10	7	4.4
5th week...	Living, autogenous	10	4	10	6	3.8

*There are 40 milking cows in this herd.

TABLE IV—*Data on vaccine treatment in herd D.**

TIME OF TREATMENT	VACCINE USED	VACCINE USED (CC)	COWS INFECTED		QUARTERS INFECTED	
			No.	%	No.	%
Beginning..	Living, polyvalent, herd-autogenous	8	11	52.4	28	33.3
1st week....	Living, polyvalent, herd-autogenous	10	11	52.4	26	30.9
2nd week...	Living, polyvalent, herd-autogenous	12	7	33.3	14	16.7
3rd week...	Living, polyvalent, herd-autogenous	12	5	23.3	9	10.7

*There are 21 milking cows in this herd.

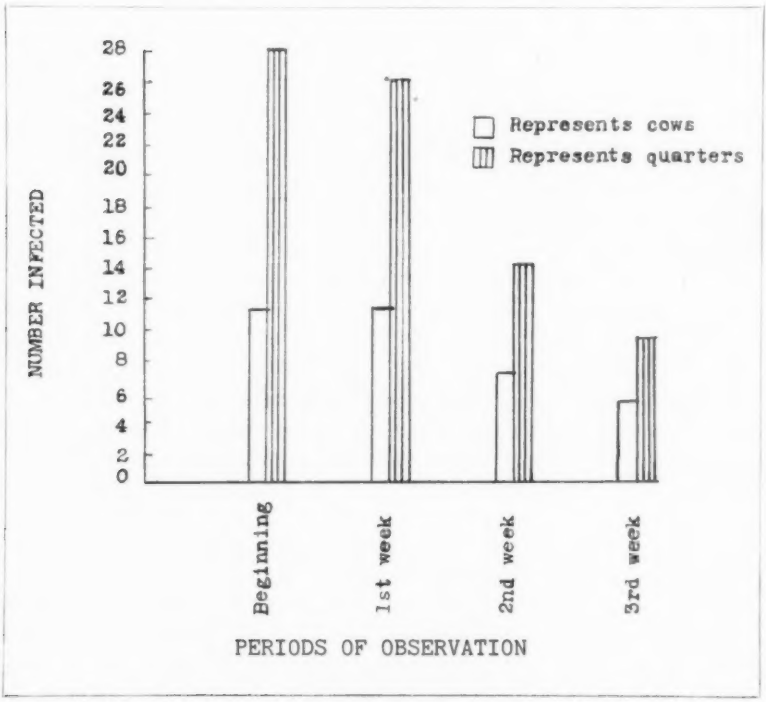

FIG. 1. Graph showing results obtained in herd D (21 milking cows in herd) by the use of living autogenous vaccine.

Tables I, II, III and IV give the results obtained in four herds picked at random from those under study. The number and percentage of cows involved and also the number and percentage of quarters involved at the weekly examinations are indicated. In interpreting the data on percentage of cows and quarters involved, one must bear in mind that there is a possibility of having four times the number of quarters involved as cows. Furthermore, in reporting the results on a herd, all animals and quarters involved are counted whether they are of the acute or chronic type of mastitis. In a study of the graph in figure 1, it is noted readily that there is a decrease in both the number of cows and also the number of quarters affected. This decrease begins soon after the second injection and continues until there are no more cases clearing up. The graph and tables show that there can not be any conclusion drawn as to whether the number of involved cows decreases faster than the number of quarters, or vice versa. It would seem that the extent of disease and the response of the animal to the vaccine treatment determines the results obtained. It is noted readily that the vaccine does not cure every case of mastitis, but the halting of the spread of the disease in the herd and the great decrease in cases involved indicate that it is of considerable value as a form of treatment.

Lacto-Vaccine

The second type of vaccine studied was essentially a lacto-vaccine. The technic in preparing this lacto-vaccine and some theoretical considerations will be given. In the bacteriological study of the milk, by culturing the milk of each quarter separately, the infected quarters in a herd were located readily.

A herd-autogenous or an animal-autogenous vaccine, depending upon the number of cases to be treated, then was prepared by aseptically milking some of the milk containing streptococci from the infected quarters into a sterile glass bottle. The amount of vaccine prepared each week depended upon the number of cases that were treated. To the streptococcus-infected milk was added a 1 per cent (1:100 dilution) aqueous solution of either gentian violet or brilliant green, so that the final dilution of the dye in the milk was 1:10,000. Adding dye (1 per cent solution) at the rate of 0.1 cc to 10 cc of infected milk gives the proper dilution of 1:10,000. The dye and milk were mixed thoroughly and allowed to remain in contact 30 minutes, with occasional shaking before the lacto-vaccine was used for injection.

There is a possibility that brilliant green in time may lose some of its specificity for certain organisms and thus gentian violet, which does not exhibit this loss, perhaps is the more desirable dye to use. The injections were made subcutaneously, immediately posterior to the point of the withers, at weekly intervals; the number of injections was decided by the progress of the disease. The size of each injection can be determined best by the veterinarian, bearing in mind the size and condition of the cow; nevertheless increasing weekly injections of 10, 15 and 20 cc are recommended.

In the bacteriological examination of the milk, at the time of detection of mastitis, it was noted that infected milk contained thousands and sometimes millions of organisms per cubic centimeter. This number of organisms was deemed sufficient for a vaccine. In the preparation of the vaccine, contamination was guarded against by using aseptic precautions in obtaining the infected milk. The added dye killed the gram-positive organisms of the normal udder flora; the streptococci were not killed, since they could be cultured from the milk after the 30-minute period of contact and for several days thereafter. Thus a living vaccine was obtained with the organisms not having undergone the modification which often results upon culturing on bacteriological media. Furthermore, this lacto-vaccine is prepared fresh each week, immediately before use, and if any immune bodies are produced locally in the udder, the added benefit of these immune bodies is obtained in the subsequent injections. The injections again were made posterior to the point of the withers. Absorption of the vaccine usually took place very readily. In a few cases a slightly raised area was noted at the point of injection, but in a very short time it disappeared. In the preparation of this vaccine no attention was placed on the type of infecting streptococcus, but the streptococcus responsible for the mastitis in the cow or herd, as found present in the infected milk, was used.

Experimental Results

Table V and the graph in figure 2 give the results obtained in one herd picked at random from those in which this vaccine was used. The disease in this herd was very extensive and spreading rapidly, as evidenced by the pre-vaccination observations and by the physical change in the milk, as observed by the owner before and during this pre-vaccination period. Two months previous to the beginning of the vaccination, this herd was examined for the physical condition of the udder and milk,

TABLE V—*Data on vaccine treatment in herd E.**

TIME OF TREATMENT	VACCINE USED	VACCINE USED (CC)	COWS INFECTED		QUARTERS INFECTED	
			No.	%	No.	%
Two months previous..			3	15.8	5	6.6
Beginning..	1-10T (brilliant green)	6	10	52.6	20	26.3
1st week....	1-10T (brilliant green)	10	10	52.6	20	26.3
2nd week...	1-10T (brilliant green)	12	10	52.6	16	21.1
3rd week...	1-10T (brilliant green)	10	10	52.6	16	21.1
4th week...	1-10T (brilliant green)	20	6	31.5	9	11.8
5th week...	1-10T (brilliant green)	20	3	15.8	3	3.9
6th week...	1-10T (brilliant green)	20	1	5.3	1	1.3
7th week...	1-10T (brilliant green)	20	1	5.3	1	1.3

*There are 19 milking cows in this herd.

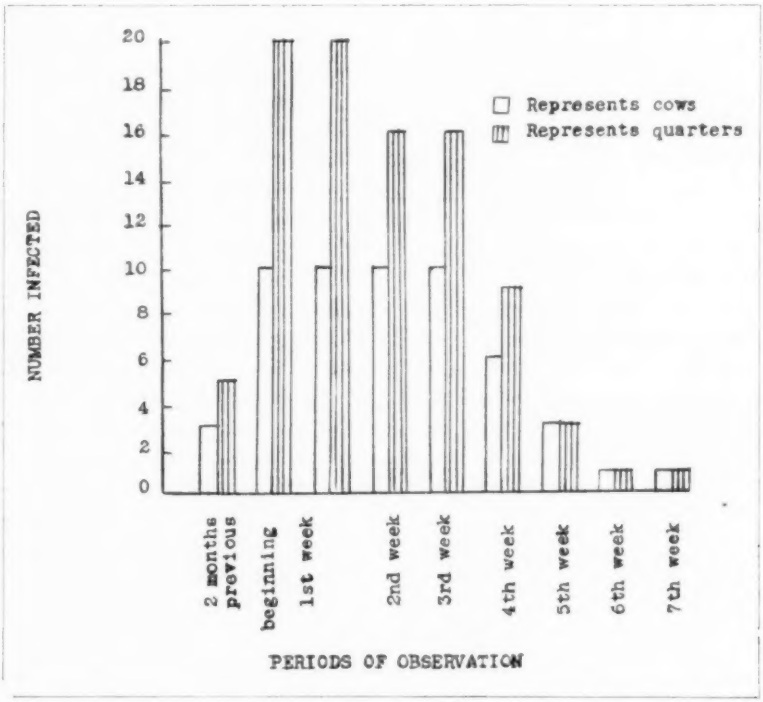

FIG. 2. Graph showing results obtained in herd E (19 milking cows) by the use of lacto-vaccine.

and the milk was examined bacteriologically for streptococci of mastitis. The results obtained are given in both the graph and table. At the time of the first examinations, the owner thought that the condition would take care of itself and consequently nothing was done. In the comparatively short period of two months, the disease had spread through his herd to the extent that over half of his cows were infected. It was at this time that vaccination was started. By the second week a decrease in infected animals was noted and it is significant that no new cases developed. This decrease continued until only one cow was involved and in only one quarter. Great damage had been done to her quarter by streptococci and staphylococci and thus it would be unreasonable to expect recovery here since it would necessitate regeneration of the udder tissue.

Discussion

The results obtained by the use of two types of vaccines on mastitis are presented herein. In presenting the data, the writer does not lose sight of the fact that there may be a spontaneous cure of the disease. Nevertheless, in the prevaccination work an actual increase in the number of cases in the herds was noted, which, with a decided decrease in the animals and quarters involved after vaccination, when no other form of medication was administered, bears evidence that the vaccines were of value in treating cases of mastitis. As before mentioned, the owners insisted that all animals be vaccinated. Thus, no control animals were used to determine the self-cure of the disease in the animals.

The data presented on all herds bear evidence that both the herd-autogenous vaccine and lacto-vaccine were very effective in safeguarding the uninfected animals from infection when the infective organisms were present in the herd. Almost immediately when vaccination was begun, no new cases developed. The duration of this protective period has not been determined definitely as yet. In all cases treated by the autogenous vaccine and lacto-vaccine, the infective streptococci in each herd were used in vaccine preparation. Thus, no definite culture was indicated as having superior value as compared to other cultures. Results show that living cultures were harmless when used as a vaccine for subcutaneous injection.

In the herds under study, all the animals of lactation age were vaccinated; this included both pregnant and non-pregnant animals. It is possible that the time of vaccination in the lacta-

tion period may bear some relation to the benefit derived. This possibility is being investigated further.

A study of the data will reveal readily that vaccines were not 100 per cent effective, but the results here presented show that good results were obtained by the use of a vaccine in preventing the spread of the disease and also in curing cases of mastitis. In certain aggravated chronic cases, where the udder tissue has been damaged greatly, we can not expect recovery; yet such cases must be included when reporting the results on the value of vaccines in mastitis. Immediate slaughter of such chronic cases is recommended, except in the instance that the animal is to be kept for her breeding value. Such a cow should be isolated properly and handled to prevent the spread of the infective organisms.

SUMMARY

1. Vaccines of the autogenous type as well as lacto-vaccines were of value in treating and obtaining recovery from infectious mastitis, as evidenced by bacteriological examination of the milk and physical examination of the milk and udder. In the few cows which did not recover, a large amount of damage had been done to the udder before vaccination. The effectiveness of the vaccine seems to depend on the extent of disease and the response of the animal to the vaccination.

2. The gentian-violet or brilliant-green lacto-vaccine may be prepared cheaply and very easily by the practitioner. The technic of preparation is outlined fully.

3. A definite protection was afforded non-infected animals in an infected herd by both the autogenous vaccines and lacto-vaccines. Three weekly injections of vaccine were given to these non-infected animals. All animals of lactation age, both pregnant and non-pregnant, were vaccinated safely.

4. The living-culture vaccines can be used with safety, since no localized infections in the udder resulted after vaccination.

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THE VALUE OF KAMALA AS A TENICIDE FOR YOUNG TURKEYS*

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The mortality from teniasis is relatively high in turkeys from one to two months of age. It is during this period that the owner becomes acutely aware that something must be done to alleviate the condition. Considerable research work has been done with kamala as a tenicide for chickens. However, only a limited amount of controlled work on the treatment of turkeys with this drug has been published.

Hall and Shillinger[1] concluded that kamala in 15-grain doses was a satisfactory drug for the removal of tapeworms in chickens. This was further substantiated by the independent work of Beach and Warren.[2] More recently, Rebrassier[3] concluded that kamala in doses of 7.5, 10 and 15 grains was not an effective tenicide for chickens. His experiment birds were treated with kamala containing not more than 5 per cent of ash, and were held for two weeks or more before being killed.

Hall and Shillinger[1] did a limited amount of experimental work to determine the tenicidal value of kamala for turkeys with apparently satisfactory results, but they state that further work is necessary to determine its dosage and prove its efficiency conclusively. Beach[4] mentions that kamala proves decidedly toxic to turkeys in many cases. He states that for this reason a few birds should be treated previous to administering the drug to the entire flock. Cram[5] has stated that turkeys and chickens that are affected with complicating diseases should not be treated with kamala because of the resulting high mortality.

The experimental work herein reported was carried on in an effort to determine the safety and efficiency of kamala as a tenicide for young turkeys.

The 63 turkeys which were used in the following experiment were obtained from eleven different flocks in which a previous diagnosis of teniasis had been made. These turkeys thus represented typical clinical cases from flocks in which losses had been experienced.

PROCEDURE

The kamala used was obtained from two different sources, one lot in tablet form, which was administered to 56 of the birds, the

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TABLE I—*Data on turkeys treated with kamala.*

TURKEY	WEIGHT (OUNCES)	DOSE (GRAINS)	FECAL EXAM. (T)	AUTOPSY FINDINGS			
				BIRDS WHICH DIED		BIRDS KILLED AFTER 2 WEEKS	
				T	C	T	C
1A	6	1.00	−			+	−
2A	7	2.00	−	+	−		
3A	5	0.25	−	−	−		
4A	6	0.50	−	+	−		
5A	6	0.25	+			−	−
6A	4	0.50	+	−	−		
7A	5	1.00	+	−	−		
8B	18	5.00	+			−	−
9B	13	1.00	+	+++	+		
10B	12	0.50	−	+	+		
11B	20	5.00	+	−	+		
12B	12	1.00	−	−	−		
13B	12	0.50	−	+++	++		
14B	14	2.00	−	−	+		
15B	12	2.00	−	−	+		
16C	24	5.00	−			++	−
17C	20	2.00	−			−	+
18C	16	1.00	+	−	−		
19C	5	1.00	−			−	−
20C	6	1.00	−			−	+
21C	4	0.50	+	−	−		
22C	5	0.50	−			+	+
23C	6	1.00	−			−	+
24C	4	0.50	−	−	+		
25D	5½	1.00	−	++	++		
26D	6	1.00	−	++	++		
27E	2½	0.25	−	+	++		
28F	15	1.00	−			−	−
29F	13	1.00	−	+	+		
30F	16	1.00	−	++	−		
31F	12	1.00	−	++	−		
32F	16	0.50	−	+	+		
33F	12	0.33	−	++	−		
34F	11	0.33	−	−	−		
35F	16	2.00	−	−	−		
36F	15	0.25	−			+++	−
37F	16	0.67	−			++	−
38G	18	1.00	+	++	+		
39G	26	2.00	−	+	+		
40G	25	0.50	−	++	++		
41G	28	1.00	++	++	++		
42G	28	1.00	−	++	−		
43G	25	2.00	−	++	−		
44G	24	2.00	+			+++	+

TABLE I—*Data on turkeys treated with kamala—Concluded.*

TURKEY	WEIGHT (OUNCES)	DOSE (GRAINS)	FECAL EXAM. (T)	AUTOPSY FINDINGS			
				BIRDS WHICH DIED		BIRDS KILLED AFTER 2 WEEKS	
				T	C	T	C
45H	20	0.50	−	+++	+		
46H	19	1.00	−	++	−		
47H	34	2.00	−			−	−
48I	32	0.125	−			+++	−
49I	28	0.25	−			−	−
50I	37	0.50	+			+	−
51I	32	2.00	+			+	−
52J	54	7.00	−			−	+
53J	51	7.00	−			−	++
54J	60	4.00	+			+	++
55J	80	12.00	+			−	++
56J	50	5.00	+			+	−
57J	26	3.00	+			−	+++
58J	41	10.00	+++			++	−
59J	62	8.00	+++			−	−
60J	44	3.00	+++			+	++
61J	71	12.00	++			+++	+
62J	41	3.00	+			−	−
63J	54	4.00	+++			+++	++

Key: T = tapeworms. C = coccidia.
− = few. ++ = numerous. +++ = very numerous.
A, B, etc. = various flocks.

other in powdered form, which was administered to the seven other birds. The total ash content of the tablets was 15.84 per cent. The ash content of the powder was 9.33 per cent. The dosage of kamala varied from $\frac{1}{8}$ to 20 grains, depending upon the size of the birds.

All feed was withheld for 15 hours from all the turkeys identified by the letters E, F and J (table I) before being treated. The feed was not withheld from the remainder of the birds previous to treatment. After the kamala was administered, the birds were kept in a clean, insect-proof room.

The birds which died following treatment were examined immediately. The birds which survived the treatment were held for a period of two weeks before being killed and examined. The droppings were collected in pans of normal saline solution, from 3 to 20 hours following treatment. Small quantities of this material were diluted further with normal saline and examined in a moist chamber against a black background. Tapeworms with heads and long chains of united segments were reported as tapeworms, single or short chains of segments being ignored.

Turkeys which died following treatment and those which were

killed after two weeks were examined for both tapeworms and coccidia. The intestines were removed and slit open in the moist chamber. The mucus and fecal material were washed from the mucosa by gentle agitation in normal salt solution. By changing the salt solution several times, all of the adherent débris could be removed from the mucosa. The intestines, as well as the diluted fecal material, were examined for tapeworms. The presence of coccidial oöcysts in the duodenum and cecums was determined by the sugar concentration method.

In considering the following results, it should be noted that kamala proved relatively harmless when administered to young normal turkeys in doses comparable to those reported in table I.

TABLE II—*Summary.*

	Birds Which Died	Birds Killed After 2 Weeks	Totals
Turkeys treated	33	30	63
Total with tapeworms	26	21	47
Tapeworms in droppings	8	15	23
All tapeworms passed	5	6	11
Total with coccidia	17	13	30
No coccidia or tapeworms	4	4	8

Turkeys That Died Following Treatment

Of the 63 turkeys treated, 33 died a few hours following treatment. (See table II.) These birds weighed from 2.5 to 28 ounces (average 14 ounces) at the time of treatment. The dosage of kamala administered to this group ranged from 0.25 to 2 grains (average 0.99 grain). The mortality was confined to the first 47 birds (1A-47H, table I), which represent the smallest turkeys. The birds first showed symptoms of prostration, followed by a semi-comatose condition and finally death. The most pronounced lesion found in these turkeys was an acute enteritis. This was most severe in the duodenum and rapidly decreased in the posterior portion of the bowel.

A total of 26 turkeys were infested with tapeworms; fourteen of this number revealed the presence of coccidia also. Eight birds passed tapeworms following treatment, while only five of these passed all of the tapeworms. Four turkeys were free of both coccidia and tapeworms.

Seventeen birds revealed the presence of oöcysts, which were found most frequently and in the greatest numbers in the cecums.

It should be noted in this connection that 13 of the 30 birds that survived treatment also revealed the presence of oöcysts.

In table I, birds numbered 1A to 7A were free of coccidia, although five died following treatment. It is interesting to note that the owner of the flock from which these birds were obtained treated the remainder of the flock with 0.5-grain doses of kamala with a resulting 70 per cent mortality.

Birds That Survived Treatment

Thirty birds survived the treatment. These weighed from 5 to 80 ounces (average 32 ounces) at the time they were treated. Th amount of kamala administered to each bird ranged from 0.125 to 12 grains (average 2.5 grains). Thus the birds which survived averaged 2.3 times as heavy as the birds that died following treatment and received 2.5 times as much kamala.

Twenty-one of these turkeys were infested with tapeworms, six of which also revealed oöcysts. Fifteen birds eliminated tapeworms following treatment, while only six of these passed all the tapeworms. Only four birds were free of both tapeworms and coccidia.

The birds which survived were not clinically affected by the presence of either tapeworms or coccidia, as is shown by the fact that they gained an average of 6.7 ounces per bird during the two weeks. The older birds apparently were able to withstand the effects of the teniasis better than the younger turkeys. Thirteen of these birds revealed the presence of relatively large numbers of oöcysts.

Samples of the tapeworms from the turkeys that survived were identified by Dr. Myrna F. Jones, of the Zoölogical Division, U. S. Bureau of Animal Husbandry, Washington, D. C., as *Metrolias-thes lucida, Hymenolepis carioca, Choanotaenia infundibulum,* and *Raillietina cesticillus.*

Conclusions

1. Kamala in doses of 0.25 to 5 grains did not prove to be a safe tenicide for turkeys weighing from 2.5 to 28 ounces.

2. Kamala did not prove to be an efficient anthelmintic for the removal of tapeworms in turkeys weighing from 2.5 to 80 ounces.

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PRODUCTION OF IMMUNITY TO CANINE DISTEMPER BY SERUM CONCENTRATE-LIVING VIRUS, SIMULTANEOUS INJECTIONS*

Preliminary Report

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With the Coöperation of
WM. J. LENTZ, *Philadelphia, Pa.*
Director of the Small-Animal Clinic
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Perfection of a concentrated, refined serum which will prevent distemper in dogs, when the living virus of Carré is injected simultaneously, on opposite sides, is announced.

Not only will the subcutaneous injection of the serum successfully hold and resist the living virus, but its simultaneous, intradermal injection on the opposite side of the body is a fundamental part of the inoculation. A single simultaneous injection of the serum and the living virus has been found to produce a complete and solid immunity. Unexposed puppies as young as six weeks can be so immunized without the necessity of a return visit. The impurities and excess protein of the serum being removed by its fivefold concentration and by a refining process, the possibility of serum shock, protein reaction or malaise is reduced.

All young dogs entering hospitals where distemper is present can be protected by administering the serum concentrate alone. Following injection, the inoculated dogs can be given the freedom of the distemper ward. The completeness of immunity in such cases has been proved by injection of the living virus three months after the dogs were treated. They continue well and free of the disease. All this demonstrates conclusively that isolation against distemper in quarantine wards is no longer necessary when susceptible animals are injected, as routine procedure, upon entering the hospital.

Young puppies can be entered in hospitals, wormed, operated upon and otherwise treated, and immunized against distemper at the same time.

The protection offered by the serum is immediate, owing to the antibodies contained in the serum, while a vaccine can only stimulate the production of antibodies in the body of the animal.

The necessity of standardization of the serum concentrate or of any serum is realized to be most important. This is being

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definitely worked out in the only way that seems practical and accurate. The laboratory methods, namely the complement-fixation and precipitation quantitative tests, are inaccurate and unreliable, in that positive reactions to these tests are not true measures of potency of the serum. Poor serums with low potency will give reactions as well as serums of high potency. The amount of serum capable of resisting the usual dose of the living virus has been determined on dogs. A short, rapid method of titrating each batch of serum produced is now being worked out.

It has been found that there is a method of preserving the living virus for such a time period that any question of its virility, after having been shipped from distant points or after having been stored for weeks, is removed. The final report will show tests of the virus to this effect.

For the past year an extensive research has been carried out as to the causal factor of distemper. As far as we know, there is no positive evidence to controvert the findings of Carré that the disease is caused by a filtrable virus.

There is not sufficient space for discussion of the technical phases of the hyperimmunization of the immune dogs other than to state that we have proved that the single massive injection of the living virus, followed by bleeding a week or so later, does not produce a serum sufficiently potent to prevent the living virus from producing distemper when injected simultaneously with the living virus. Serums prepared in this manner failed to prevent death in ferrets when subcutaneously injected simultaneously with the subcutaneous injection of the living virus, whereas those serums prepared by our method of hyperimmunization and concentration showed a 50 per cent survival.

It will suffice to state that no detail will be omitted in the final report in reference to the preparation of the serum, with the view of retaining its highest potency and its greatest yield by any method of concentration. Cultures of the *Bacillus bronchisepticus* and various streptococci recovered from virus spleens also are injected periodically to insure a passive protection against the secondary invaders, considered by some investigators to be the incitant factor as well as the cause of death. The concentrated serum in the dilution of 1:400 will agglutinate *B. bronchisepticus*, and in dilution of 1:600 will agglutinate the *Streptococcus cerebritis canis*.

After all, the motive of the entire work is to produce not an experimental serum but a practical one of unexcelled potency, with least volume and with its inert proteins removed. By so

doing, it will be practical from a commercial and economic standpoint.

The necessity of keeping accurate records of every case immunized by the serum concentrate-living virus method was recognized from the inception of this experiment, beginning May 5, 1932. The value and use of serum as opposed to vaccine therapy was realized many years before. Opinion and thought count for little, however, unless recorded in fact. As a result of this permanent record, we have collected, with collaborators, over 600 case records, giving a description, ownership, etc., of every dog inoculated. These records show also whether the dog was exposed or unexposed prior or subsequent to the inoculation, hospitalization and microscopic examination for worms. Observation periods were rigidly kept at 3, 6, 9, 12 and 15 months, in every case, the date of inoculation determining, of course, the length of the period of observation. The living virus (Lederle) has been employed because of its known potency, and it also has been used to produce the disease, in order to obtain active spleen virus for the hyperimmunization of the immune dogs. Our complete records of all the cases are available for inspection at any time.

Dr. George J. Goubeaud, Borough Veterinarian, Queens County, Department of Health, New York City, reports as follows:

> From August 1, 1932, to January 1, 1933, twenty-four dogs of various breeds were inoculated with the serum concentrate subcutaneously and the virus (Lederle) intradermally, on opposite sides of the body. I know of no dogs to date (August 10, 1933) that have developed distemper in this group since.
>
> From January 2 to July 15, 1933, forty dogs were inoculated simultaneously as above. No distemper developed up to date (August 10, 1933).
>
> Nineteen hospital cases received serum concentrate alone, mostly young dogs three months to a year, kept in the hospital for periods varying from two days to one week. None have developed distemper to date (August 10, 1933).
>
> One Shepherd, female, exposed in the hospital for two weeks and then given serum concentrate with virus on the opposite side, developed convulsions and evidence of distemper and died three weeks after the injection.
>
> Total number of cases treated—84 (July 15, 1933). No dog developed distemper with the exception of the last, exposed, experimental case.

The following report covers dogs treated at the Small-Animal Clinic, University of Pennsylvania Veterinary School, Philadelphia, Pa., under the direction of Dr. Wm. J. Lentz:

> I. From January 25 to April 20, 1933, eighteen dogs considered not to have been previously exposed, received 4 cc of serum con-

centrate subcutaneously, followed five days later by 4 cc of serum concentrate and 0.25 cc of virus (Lederle) intradermally. All of these dogs were confined to the hospital during the immunization. Eradication of worms and fleas, treatment of some for catarrhal enteritis, rickets, one fracture and two ovariotomy operations proceeded at the same time. All of these dogs have remained normal to date.

II. Eighty-two dogs were classified as exposure being possible. Thirteen developed pneumonia and died. Eleven of those remaining were confined to the hospital during the immunization period (4 cc of serum concentrate subcutaneously and, five days later, 4 cc of serum concentrate and living virus as in the first class). The remaining animals were members of one group treated outside of the hospital under exposure to adverse weather conditions and poor housing.

III. From April 29 to June 1, 1933, eighteen dogs displayed positive symptoms of distemper, with either bronchopneumonia or enteritis, or both, present in 14 of these cases. Eight of these animals received 8 cc of the serum concentrate as an initial dose, followed three days later by 4 cc. Four dogs died with pneumonia complications; 14 recovered.

IV. From March 25 to August 10, 1933, two dogs described as a 3-month-old cur and a 5-month-old Dalmatian, found not to have been exposed previously to distemper, were entered in the University of Pennsylvania Veterinary Hospital, on March 25, 1933. Two weeks after receiving 4 cc of concentrated serum (without living virus), they were placed in the distemper ward, remaining there ever since (to August 10, 1933). On July 6, 1933, both of these animals received 0.25 cc of living virus (Lederle) subcutaneously alone. On August 1, 1932, a second dose, as above, of living virus was injected subcutaneously. There has been no evidence of distemper to date and they will remain in the distemper ward where diseased animals always are present.

Dr. Lentz goes on to say:

To date (August 10, 1933), reports on the simultaneous serum concentrate-living virus method of immunizing dogs against distemper, as shown by the records I have kept, on the whole are very convincing.

I am led to believe that this method of immunization, comparable in principle to that so successfully employed in combating hog cholera, will meet with the same success and be used universally by the profession.

My favorable reaction, aside from the actual results obtained to date, is that the side to side simultaneous use of the serum and the living virus requires usually but one visit, thus reducing the cost to the owner of the dog. Furthermore, the relatively small dose of the serum, concentrated as it is, has obvious advantages for hypodermic medication.

Heretofore, I have been somewhat reluctant in advising medication or inoculation, feeling that the whole question was still in the experimental stage. Moreover, I have frequently made the statement that successful immunization against distemper would be possible only when a sufficiently potent antiserum was used in conjunction with the unattenuated living virus.

Dr. Victor Ross, Bureau of Laboratories, Department of Health, New York City, reports as follows on the experiments performed with ferrets:

These experiments were done with the object of learning whether the concentrated serum (Little) would "hold" the virus of canine distemper when injected side to side in the ferret. The data will be published in more detail later. Only the deaths and survivals will be given here, with little comment.

All injections were made subcutaneously. Of six control ferrets receiving the canine virus alone, five died. Of four ferrets receiving the virus and 20 cc of purchased, unconcentrated serum, side to side, all died. Each of the four was injected with a different serum. Of four ferrets receiving the virus and 4 cc of concentrated serum, two died (delayed deaths), another had the disease and recovered, and one remained well throughout. All these animals were kept in a single room.

There also were three ferrets, each of which received 4 cc of concentrated serum, followed, two days later, by 4 cc of concentrated serum and the virus, side to side. These animals were kept in a different building but were handled by the same person who handled the remaining ferrets. They remained entirely well for a period of over two weeks, at which time they were transferred to the room containing the larger group of ferrets, a number of which were very ill at this time. The three ferrets contracted the disease and died. The anomalous result of 4 cc holding the virus in two out of four cases and of 8 cc not holding in any of three may possibly be explained as follows: The canine virus does not immunize ferrets. The 4 cc partially holds the canine virus, and the animals, being exposed from the very beginning to ferrets which are coming down with the disease, become gradually immunized to what may now be considered the ferret virus. The 8 cc also held the canine virus which was injected, but the ferrets had not the benefit of the gradual exposure to the disease. When brought suddenly into contact with the disease, they contracted it and died.

ACKNOWLEDGMENT

Grateful acknowledgment is made to Dr. Victor Ross, Department of Bacteriology, New York University; Abraham J. Klein, Bureau of Laboratories, New York City, and Dr. George J. Goubeaud, Bayside, L. I., for their kind assistance.

Smallpox Vaccine from Chicken Eggs

A method of producing smallpox vaccine from chicken eggs, instead of from calf lymph, has been achieved by Col. W. D. H. Stevenson and Dr. G. G. Butler, of the British Government Lymph Establishment, according to a report in *Science* for August 4, 1933. This method is said to be a modification of the technic developed by Mr. A. M. Woodruff and Prof. E. W. Goodpasture, of Vanderbilt University, Nashville, Tenn.

The report quotes *The Lancet* as saying that the new method opens the possibility of large-scale production of a bacteria-free virus for vaccination; that it is not so arduous or expensive as the production of calf lymph, and that the yield is excellent.

OBSERVATIONS ON THE MACROSCOPIC DIAGNOSIS OF FOWL PARALYSIS. Erwin Jungherr. Poultry Sci., xii (1933), 3, p. 184.

In view of the complex etiology of paralytic conditions in birds, the recognition of the underlying disease factors is of utmost importance. True fowl paralysis, range paralysis or *neurolymphomatosis gallinarum* is a definite disease entity. The disease probably is not contagious in nature but certain evidence points toward a transmission of the inciting factor through the egg. With this possibility in mind, the diagnosis of true fowl paralysis plays an important part in any contemplated program of eradication. The only possible way of diagnosis is the examination of the nervous system by pathological and histopathological methods. Of the two methods the microscopic study is preferable but the gross examination of the sciatic trunk and brachial plexus is serviceable in routine diagnostic work and should be an established procedure. The gross examination should be conducted on at least four birds from each suspected lot. Nerve lesions consisting of changes in size and consistency of the nerve tissue are most apt to be found in the dorsal root ganglia of one or the other side of the brachial plexus.

STUDIES ON THE ETIOLOGY OF "RUPTURED YOLK." H. A. Hoffman. Poultry Sci., xii (1933), 3, p. 189.

The examination of field cases from 87 flocks suffering from the condition popularly known as "ruptured yolk" revealed the fact that 48.2 per cent were infected with *P. avicida*. Lesions somewhat similar were found in birds from a few flocks from which *S. gallinarum* or *S. pullorum* were recovered. In 24 per cent of the cases bacterial infections were not identified. The gross lesions in field cases from which *P. avicida* was recovered resembled those usually described for fowl cholera. In addition to these lesions, definite changes were found in the ovaries of many of the birds and the ova often ruptured or the yolk material escaped without a definite break in the yolk membrane. The changes in the ovary with the accompanying loss of yolk

material into the abdominal cavity are closely related to the course of the disease. The birds which died suddenly failed to show definite changes in the ovaries. "Ruptured yolk" is not due to the rupture of otherwise healthy ova. The fact that the disease may occur in flocks producing very few eggs suggests that it is not due to high production. Bacteria are probably the most common cause of "ruptured yolk."

STUDIES ON COCCIDIOSIS. IV. Mortality and infection among artificially infected chickens. Roy L. Mayhew. Poultry Sci., xii (1933), 3, p. 206.

Data are presented on the number of deaths and cases of hemorrhage occurring among inoculated chickens during the period of illness from coccidiosis. When raised under closely controlled conditions, the total number of deaths was 152 out of 671 inoculations of birds (22.7 per cent). The results are summarized from a series of inoculations made on 47 different dates and extending over a period of three years. The distribution of mortality by days also is given. The largest number of deaths occurred on the sixth day after inoculation, when 65 (42.7 per cent) died. It is believed that the deaths occurring after the eighth day were due largely to other complicating factors, chiefly temperature changes. The high percentage of infection without hemorrhage suggests the importance of application of sanitary measures by the flock-owner in epizöotics of coccidiosis.

ACTINOBACILLOSIS OF CATTLE IN THE UNITED STATES. Luther Thompson. Jour. Inf. Dis., lii (1933), 2, p. 223.

Actinobacillosis is common among cattle in the United States. The condition here is similar to that in other countries where the greater percentage of the so-called bovine actinomycosis is due to *Actinobacillus lignieresi*. The condition known as "wooden tongue" is said by European investigators to be due entirely to this organism. One case of this kind in the present study yielded a culture of Actinobacillus. Most cases of involvement only of the cervical glands were due to Actinobacillus. The few cases of infection of bone encountered were due to Actinomyces. Both Actinobacillus and Actinomyces produce the aggregations known as "sulfur granules." The quickest way of distinguishing the two organisms in pus is by staining the crushed granules by Gram's method. Actinomyces granules stained in this way show

numerous Gram-positive, rod-shaped forms, whereas the Actinobacillus granules give no Gram-positive organisms, but with careful search a few small Gram-negative bacillary forms usually may be found. The only points of similarity between the two organisms are the ability to produce similar lesions in cattle and the ability to produce sulfur granules. The granules formed by Actinobacillus usually are less conspicuous and often can be found only with the aid of the microscope. Agglutinations and absorption of agglutinins indicate that there is variation among strains of Actinobacillus as to antigenic structure.

STUDIES ON MAGNESIUM DEFICIENCY IN ANIMALS. III. Chemical changes in the blood following magnesium deprivation. H. D. Kruse, Elsa R. Orent and E. V. McCollum. Jour. Biol. Chem., c (1933), 3, p. 603.

Restriction of animals to a ration containing only 1.8 parts per million of magnesium, but adequate amounts of other constituents, leads to a syndrome previously designated as magnesium tetany. There is an early and progressive decrease in the magnesium content of the serum. Shortly thereafter there is a marked increase in total cholesterol with a commensurate decrease in fatty acids so that the total fat remains constant. These changes prevail until death. Terminally the non-protein rises. No other blood constituents undergo alteration. The authors suggest that these blood changes may be related to the symptoms of magnesium deficiency. The lowered magnesium concentration in the serum is considered in conjunction with tetany, while the lipoid changes are taken as indicative of nutritive failure. The typical chemical changes in the blood in magnesium tetany offer no points of similarity to other types of tetany.

BRONCHIAL DISINFECTION AND IMMUNIZATION. The effects in rabbits of intrabronchial injections of various chemical disinfectants. John A. Kolmer. Arch. Int. Med., li (1933), 3, p. 346.

The principles of chemotherapeutic disinfection by intrabronchial treatment with chemical agents are discussed; likewise, the possible changes and contraindications to bronchial disinfection by the local or topical application of chemical agents by bronchial lavage. A large number of disinfectants have been studied for bactericidal and spirocheticidal activity *in vitro*, in a mens-

truum rich in protein, for the selection of compounds for bronchial disinfection. Several have been found suitable. Intratracheal injections of iodized oil in rabbits followed by roentgenographic examinations have shown that it is possible to secure a wide intrapulmonary distribution of solution of chemical disinfectants by this route of administration. The toxicity of a large number of chemical disinfectants has been determined in rabbits by intratracheal injections of 1 cc per kilo of weight every two days. The approximate maximum tolerated doses of from 6 to 24 injections are given. Pathologic tissue changes produced experimentally in the various portions of the respiratory tract and kidneys of rabbits is presented. Repeated intratracheal injections of various disinfectants in bactericidal concentrations may be given with no or with slight injury to the bronchi and lungs of rabbits.

STUDIES ON THE SURVIVAL TIME OF BOVINE TUBERCLE BACILLUS IN SOIL, SOIL AND DUNG, IN DUNG AND ON GRASS, WITH EXPERIMENTS ON THE PRELIMINARY TREATMENT OF ORGANIC MATTER AND THE CULTIVATION OF THE ORGANISM. C. G. Maddock. Jour. Hyg., xxxiii (1933), 1, p. 103.

Some virulent *Bacillus tuberculosis* can survive a period of six months of exposure in soil, in soil and dung mixtures, and in dung. Since the results thereafter were consistently negative, it appears that in the south of England, exposure for a period of about seven months (June to December, inclusive) suffices to kill *B. tuberculosis* in soil, in soil and dung mixtures and in dung. In spite of exceptionally heavy rain after infection of grass, followed by great heat and dryness, all of the plots gave positive results after exposure for 14 days and one plot after 49 days. Tuberculin tests upon surviving guinea pigs indicate that this period is likely to be extended considerably.

DO TUBERCULOUS AND NON-TUBERCULOUS SERUMS POSSESS BACTERICIDAL PROPERTIES FOR THE TUBERCULOSIS BACILLUS? O. Kirchner. Abst. Arch. Path., xv (1933), 5, p. 729.

Undiluted serum was unsatisfactory for the growth of tuberculosis bacilli, but improved markedly when properly diluted. There was no evidence of specific bactericidal properties for the tuberculosis bacillus in 32 tuberculous and 20 non-tuberculous serums. The differences which were noted would be explained on the basis of physico-chemical and nutritive factors.

EFFECT OF LIGHT ON THE BACTERICIDAL PROPERTIES OF SERUM. Alfred Pettersson. Abst. Arch. Path., xv (1933), 5. p. 733.

The ultraviolet rays destroyed the bactericidal properties of serum much more effectively than did the visible rays. The complement was considerably more sensitive than the bacteriolysins and the bactericidal products of leukocytes.

WATER METABOLISM IN PULMONARY TUBERCULOSIS. M. Keresztes. Abst. Arch. Path., xv (1933), 6, p. 851.

In pulmonary tuberculosis retention of water occurs without the production of noticeable edema. The cause of this is not a disturbance of the circulation but faulty metabolism. Water retention is more likely to occur in exudative malignant types of the disease.

BIOLOGY OF THE TUBERCLE BACILLUS. III. Does the so-called fatty capsule serve to protect the tubercle bacillus? John Weinzirl. Jour. Bact., xxv (1933), 5, p. 447.

When *Mycobacterium tuberculosis* (H 520, apathogenic) was grown upon a nutrient medium containing 5 per cent glycerol, it developed approximately 24 to 32 per cent of lipins; on the same medium without glycerol, it developed about 5 to 9 per cent of lipins. When the fat and lean bacilli were exposed to chemicals and to physical agents more than half of the trials showed no difference in resistance between the two cultures. When a difference was shown, the resistance was slightly greater in case of the fat bacilli. The slightly greater resistance shown by the fat bacilli may have been due to their growth on a more favorable medium. The author concludes that the results tend to show that the so-called fatty capsule does not materially increase the resistance of the bacilli to chemical and physical agents.

THE GERMICIDAL EFFICIENCY OF SODIUM HYDROXID. Ernest C. McCulloch. Jour. Bact., xxv (1933), 5, p. 469.

Sodium hydroxid in the form of high-test commercial lye appears to satisfy the needs for a disinfectant which is suitable for use in barns and stables and which is inexpensive, odorless and very efficient against *Brucella abortus* and related microorganisms. The velocity of disinfection with sodium hydroxid between 25 and 2° C. is independent of the temperature changes. This phenomenon appears to be correlated with the peculiar

physico-chemical reaction of sodium hydroxid solutions to temperature changes, wherein the ratio of the hydroxyl-ion activity to the hydrogen-ion activity increases with a decrease in temperature. The increase of the hydroxyl-ion activity over the hydrogen-ion activity at the lower temperature approximately balances the greater germicidal efficiency of each hydroxyl-ion at the greater temperature. At a given temperature, a given hydroxyl-ion concentration possesses a germicidal activity which is independent of the amount of alkali used to attain the hydroxyl-ion concentration. The hydroxyl-ion is the important factor in the destruction of microörganisms of sodium hydroxid.

MODIFICATION OF THE PATHOGENICITY OF PSEUDORABIES VIRUS BY ANIMAL PASSAGE. Richard E. Shope. Jour. Exp. Med., lvii (1933), 6, p. 925.

Pseudorabies virus, Iowa strain ("mad itch") after passage through guinea-pig brain, fails to produce infection in guinea pigs when injected subcutaneously unless enormous doses are employed. Such virus is still pathogenic for rabbits when given subcutaneously and for rabbits and guinea pigs intracerebrally. Comparisons of the amounts of virus present in the brains of rabbits and guinea pigs following fatal cerebral infection shows that the latter contain, per gram, only one-tenth the amount of virus in the former. Comparing the resistance of the two species to subcutaneously administered pseudorabies virus, it has been found that rabbits are approximately 100 times more susceptible than guinea pigs. Over and above the working of these two factors, guinea-pig passage appears to achieve some actual attenuation of virus tested by subcutaneous inoculation into guinea pigs.

EFFECTS OF LOW TEMPERATURES UPON ENCYSTED TRICHINELLA SPIRALIS. Donald L. Augustine. Amer. Jour. Hyg., xvii (1933), 3, p. 697.

Raw pork loin roasts, in which trichinosis muscle had been inserted, were rapidly brought to low temperatures varying from −18.1 to −34.6° C., recorded by means of thermo-couples inserted into the trichinosis muscle itself. Temperatures as low as −21° C. produced no demonstrable injury to the parasites, whereas reduced vitality resulted from exposure to a temperature of −27.6 C. Still greater injury was demonstrated on larvae exposed to −30.9° C. Larvae subjected to a temperature of −33.9°

C. produced no infection in test animals, although about 12 per cent appeared normal microscopically. Complete destruction was demonstrated on trichinella larvae to have occurred at – 34.6° C. Complete destruction occurred also when the larvae were brought rapidly to a temperature of – 18° C. and held there for 24 hours. The author concludes that raw pork, in commercial quantities, may be rendered safe, as far as trichinosis is concerned, by either rapidly lowering its temperature to – 18° C. and holding for at least 24 hours or by rapidly lowering the temperature to – 35° C.

OBSERVATIONS ON THE BACTERIAL FLORA OF SOME SLAUGHTER HOUSES. R. B. Haines. Jour. Hyg., xxxiii (1933), 2, p. 165.

A study has been made of the bacterial flora of the air, walls, floor, and swabbing water of two types of slaughter-houses, type I the small slaughter-house and type II the large abattoir. If each animal is regarded as a potential source of bacteria which will be scattered during handling, the gross infection in the large abattoir is much larger. However, the air of the large abattoir carries a much smaller load of organisms than the small slaughter-house. In the former, 9 per cent were intestinal organisms, while in the latter 19 per cent were of intestinal origin. The most striking difference was found in the degree of infection of the water used for swabbing the carcasses. In the small slaughter-house the colon count was 5,000 per 10 cc, while in the large abattoir the count was 5 per 10 cc.

BRONCHIAL DISINFECTION AND IMMUNIZATION. I. The effect in rabbits of intrabronchial injections of vaccines, bacteriophage and antivirus. John A. Kolmer. Arch. Int. Med., li (1933), 5, p. 692.

Phagocytosis is apparently the principal mechanism of defense and recovery in chronic bronchitis, bronchiectasis and other types of suppurative pneumonitis. Any therapeutic procedure promoting phagocytosis of the streptococci, staphylococci and other organisms in the bronchial secretions of suppurative pneumonitis will likely prove of therapeutic value. The administration of autogenous vaccines and antiviruses by intrabronchial injection for the purpose of mobilizing phagocytes, stimulating phagocytosis and promoting local and general antibody production is suggested. Intrabronchial injections of antivirus may also produce direct destruction of infecting organisms in addition to pro-

moting macrophage phagocytosis and antibody production. Intrabronchial injections of bacteriophage may result in the direct destruction of the living organisms as well as stimulate phagocytosis. Such injections in doses of 1 cc per kilo of body weight of rabbits were extremely well borne. Such injections have increased the number of microphages and macrophages in the bronchial secretions of rabbits, produced an increase of opsonins and agglutinins in the blood of rabbits, and have increased their resistance to intratracheal inoculations of virulent cultures, especially in the case of type I pneumococcus.

THE INFLUENCE OF MINERAL METABOLISM UPON NEPHROTIC EDEMA. William S. Hoffman and Wilber E. Post. Jour. Clin. Invest., xii (1933), 4, p. 613.

Ingestion of water, sodium or chlorid produces a transudation of these substances across the capillary walls to keep the osmotic relations of the plasma and intracellular fluids somewhere near normal, until the excretion by the kidneys restores the normal body content. A low serum-protein concentration produces a tendency for accumulation of the extracellular fluid because of the lag in the return of fluid into the blood-stream. The extent of this accumulation is determined, among other factors, by the speed with which the kidneys carry on the excretion of water, sodium and chlorid. Adequate excretion of sodium in nephrosis apparently is possible only when the serum-sodium concentration is at a normal and higher than normal level. The serum-sodium concentration seems to be related to that of red-cell potassium and factors which raise the latter may raise the concentration of serum-sodium and thereby increase the urinary excretion of sodium.

THE MEASURED EFFECT OF LAPAROTOMY ON THE RESPIRATION. Henry K. Beecher. Jour. Clin. Invest., xii (1933), 4, p. 639.

Following laparotomy there is marked decrease in lung volumes. The effect upon residual air alone is quantitatively not significant. The same is true of maximum volume. The effect upon subtidal volume is significant—it decreases one-fifth. Reasons are given for believing that this shrinkage is due to a type of diffuse partial collapse of the lungs not heretofore demonstrated, rather than to local total collapse. Crippling of the respiratory mechanics, indicated by decrease of vital capacity, precedes the development of this diffuse type of collapse.

Regular Army

First Lieutenant Maurice W. Hale is relieved from further assignment and duty at the San Francisco port of embarkation, Fort Mason, Calif., effective upon the arrival of the U. S. Army transport "Meigs," in New York, and will proceed to Washington, D. C., and report to the commanding general, Army Medical Center, for duty.

Second Lieutenant Edgerton L. Watson is relieved from duty at Fort Myer, Va., effective on or about July 15, 1933, is then assigned to duty at the San Francisco port of embarkation, Fort Mason, Calif., and will proceed to New York, N. Y., and report to the superintendent, Army Transport Service, for duty as transport veterinarian on the return trip of the U. S. Army transport "Meigs."

Major Everett C. Conant is relieved from duty at the Presidio of Monterey, Calif., effective in time to proceed to San Francisco, Calif., and sail on the transport scheduled to leave that port for New York, N. Y., on or about August 1, 1933; upon arrival in New York will proceed to Lexington, Ky., and report to the commanding officer, remount purchasing and breeding headquarters, for duty.

The appointment and assignment of the following-named second lieutenants, Veterinary Corps Reserve, as second lieutenants in the Veterinary Corps, Regular Army, with rank from July 1, 1933, is announced:

Wayne Otho Kester, Akron, Ohio.
Robert Arthur Boyce, Jr., Philadelphia, Pa.
Clarence Leonard Taylor, Odebolt, Iowa.

They are assigned to duty at Fort Hayes, Ohio, Carlisle Barracks, Pa., and Fort Snelling, Minn., respectively.

The promotion of Second Lieutenant Austin T. Getz to the grade of first lieutenant with rank from July 22, 1933, is announced.

Veterinary Reserve Corps

New Acceptances

Hirleman, Ward Brown..Capt.....709 Academy Ave., Waynesboro, Ga.
Hagler, Curtis Edward..2nd Lt....925 Cleveland Ave., Loveland, Colo.
Hayes, Raymond William..2nd Lt..Blairstown, Mo.
Johnston, Joseph Leon..2nd Lt....Box C, Sylvester, Ga.
Hoyt, Frederick James..2nd Lt...Greene, N. Y.
LaFrance, William Joseph..2nd Lt.206 E. Lincoln St., Ithaca, N. Y.
Liner, Nelson Richard..2nd Lt....Amenia, N. Y.
Olmsted, Richard Calvin..2nd Lt..43 Greene St., Catskill, N. Y.
O'Neil, Henry Elmer..2nd Lt.....Westport, N. Y.
Sears, Richard Martin..2nd Lt....48½ W. Genesee St., Baldwinsville, N. Y.
Wohnsiedler, Herbert Gustav.2nd Lt.129 Church St.. Carthage, N. Y.
Armstrong, James William..2nd Lt.114 Elmdale Ave., Providence, R. I.

Bachtel, David Henry..2nd Lt.....R. D. No. 3, Massillon, Ohio.
Bramley, Melvin James..2nd Lt...7216 Hecker Ave., Cleveland, Ohio.
Bushnell, Fred Forbes, Jr..2nd Lt..494 E. Center St., S. Manchester, Conn.
Cameron, Bertram Novell..2nd Lt.64 School St., Everett, Mass.
Davidson, J. LaVere..2nd Lt.....2226 Oakland Drive, Kalamazoo, Mich.
Earhart, Robert Nixon..2nd Lt...342 W. 9th Ave., Columbus, Ohio.
Griffel, Raymond Richard..2nd Lt..Newell, Iowa.
Harter, William Lewis..2nd Lt....1374 N. Van Ness, Hollywood, Calif.
Henson, William Richard..2nd Lt..R. D. No. 2, Elyria, Ohio.
Larson, Emil Vernon..2nd Lt.....Evansville, Minn.
McKenzie, Carl Van..2nd Lt.....181 12th Ave., Columbus, Ohio.
McKitrick, John Leslie..2nd Lt...Station "B", R. D. No. 1, Columbus, Ohio.
Maike, Arthur Albert..2nd Lt.....R. D. No. 4, Fremont, Ohio.
Maxey, Howard Curtis..2nd Lt....Riverton, Iowa.
Omdalen, Rudolph Oscar..2nd Lt..140 W. Frambes Ave., Columbus, Ohio.
Pomeroy, Benjamin Sherwood..
2nd Lt. .....850 Linwood Place, St. Paul, Minn.
Scothorn, Marion Woolever.2nd Lt.Ashville, Ohio.
Sinek, Charles James..2nd Lt.....R. D. No. 2, Pocahontas, Iowa.
Stern, Aaron..2nd Lt.....467 Edgewood St., Hartford, Conn.
Tekse, Lloyd Christopher..2nd Lt..711 Cleveland Ave., Fergus Falls, Minn.
Thiele, Mervin Ivan..2nd Lt.....Bayard, Iowa.
Todd, Frank Arnold..2nd Lt.....Merrill, Iowa.
Todd, Guy Harold..2nd Lt.....Merrill, Iowa.

Promotions

To

Mimnaugh, John Joseph..Capt.....207 S. Main St., New Canaan, Conn.
Warmoth, Wm. Daniel..Capt.....Macon, Mo.
Currie, Arthur James..1st Lt.....Greenwood, Wis.

Transferred

Wallis, Robert Leon..2nd Lt.....Walhalla, S. C., from Inf-Res.
Schondau, Theodore..1st Lt.....15 First Ave., Halethorpe, Md., to Aux.

Rabies Quarantines

All dogs in Bloomington, Ill., were under a strict quarantine for rabies, from July 23 until August 12. After the quarantine period all dogs had to be vaccinated before being allowed to run at large.

Johnson Township, Barry County, Michigan, was placed under a 30-day quarantine for rabies the latter part of August, following the positive diagnosis of a case in a dog that had bitten three persons.

National Dog Week—October 1-7, 1933.
A Pedigreed Dog in Every Home.

OKLAHOMA VETERINARY MEDICAL ASSOCIATION

The Oklahoma Veterinary Medical Association held its nineteenth semi-annual meeting on the grounds of the United States Remount Station, Fort Reno, June 19-20, 1933.

The meeting, which was well attended and decidedly different from any other ever held by this Association, was the subject of much favorable comment by members and visitors alike. A large part of the credit for its success is due to Post Commander Col. James E. Shelly and his veterinary staff, who placed all of the facilities of the Post, including an abundance of clinical material, at the disposal of the Association, and otherwise assumed full responsibility for the entertainment and comfort of the visitors.

Dr. R. R. Dykstra, of Kansas State College, gave an instructive and interesting talk on "Problems in Equine Practice," in which he covered a wide range of practical questions that are met frequently in horse practice. Major James R. Sperry, of the Fort Reno veterinary staff, discussed "The Duties and Responsibilities of the Army Veterinarian." Capt. E. E. Hodgson, also of Fort Reno, spoke on the endocrines and their significance in veterinary medicine. This subject, although somewhat new to most veterinarians, proved to be intensely interesting and brought out many questions and a lively discussion. Dr. A. T. Kinsley, of Kansas City, Mo., appeared on the program for a discussion of swine diseases. His paper, entitled, "The Infectious Swine Disease Situation," reviewed the most important diseases met in swine practice and stressed the importance of accurate differential diagnosis. Ameboid dysentery and swine erysipelas were given special attention.

The afternoon of the first day was devoted to the large-animal clinic and to postmortem meat inspection. Demonstrations of the following were given: intravenous medication and the collection of blood samples; the intradermic tuberculin test, and nerve blocking for local anesthesia in operations about the head. Dr. J. H. Kitzhofer, B. A. I. inspector-in-charge, Oklahoma City, demonstrated the procedure used in government postmortem

meat inspection, on a number of carcasses slaughtered expressly for this purpose.

The forenoon of the second day was occupied with a paper, "Tuberculin Testing," by Dr. W. I. Bowersox, of Topeka, Kan., and a round-table discussion led by Dr. R. R. Dykstra. The meeting was closed with an unusually interesting small-animal clinic conducted by Dr. H. W. Ayers, of Oklahoma City.

Among the delightful features of this meeting were a regulation army dinner service in regular army field style, a barbecue lunch on the reservation and a banquet at the Southern Hotel, in El Reno. All three of these functions were attended by a large number of ladies, who expressed themselves as delighted with the arrangements made for their entertainment.

C. H. FAUKS, *Secretary.*

PACIFIC NORTHWEST VETERINARY MEDICAL ASSOCIATION

The Pacific Northwest Veterinary Medical Association, comprising the associations in the states of Washington and Oregon and the province of British Columbia, met in Olympia, Wash., July 10, 11 and 12, 1933. About 75 veterinarians and 30 ladies were in attendance at the decidedly successful meeting. At the special invitation of Governor Martin, of Washington, the conventioners visited him in his offices, listened to a short, informal talk and later were shown through the new $7,000,000 capitol.

Dr. R. G. Cuthbert, of Vancouver, president of the British Columbia Veterinary Association, presided at the Monday session and made the opening address. Hon. E. N. Steele, mayor of Olympia, welcomed the visitors to the city, and Dr. B. T. Simms, of Corvallis, Ore., responded. Dr. Fred W. Lange, of Salem, president of the Oregon Veterinary Medical Association, presided at the Tuesday session, and Dr. O. G. Burton, of Tacoma, president of the Washington State Veterinary Medical Association, at the Wednesday session. The following literary program was given:

"Some Experiences with Anthrax," by Dr. W. Graham Gillam, Cloverdale, B. C.

"Diseases of the New-Born Colt," by Dr. S. L. Brown, Portland, Ore.

"New Researches in the Pathogenesis of Azoturia," by Dr. Hilton Smith, Pullman, Wash.

"Meat Inspection," by Dr. K. M. Oliver, Snohomish, Wash.

"Case Reports, Small Animals," by Dr. J. K. Leaverton, Portland, Ore.

"Garbage-Feeding Troubles in Swine," by Dr. J. R. Fuller, Walla Walla, Wash.

"Caudal Anesthesia," by Dr. G. F. R. Barton, Chilliwack, B. C.

"Essentials in Developing a Live Stock Industry," by Mr. F. E. Balmer, Director of Extension, Washington State College, Pullman, Wash.

"Equine Encephalomyelitis," by Dr. J. A. Flanigan, Eugene, Ore.

"Diet of Small Animals," by Dr. G. R. Stewart, Spokane, Wash.

"Veterinary Work in South China," by Col. J. R. Shand, V. C., U. S. A., Fort Lewis, Wash.

"The Diagnosis of Gastric and Intestinal Diseases in the Dog and Cat," by Dr. R. G. Cuthbert, Vancouver, B. C.

"Publicity for the Veterinary Profession," by Dr. W. H. Lytle, Salem, Ore.

"Laboratory Diagnosis for the Practitioner," by Dr. Hilton Smith, Pullman, Wash.

A clinic was held at Fort Lewis on Tuesday afternoon, under the direction of Capt. H. R. Leighton, V. C., U. S. A., of Fort Lewis, and Dr. W. E. Long.

A dinner and dance were held at the Olympia Golf and Country Club on Tuesday evening. A feature of this occasion was the presentation of leather toilet cases to two Washington members—Dr. Carl Cozier, of Bellingham, and Dr. C. S. Philips, of Mount Vernon—in honor of 25 years of continuous membership in the Association. They are the only two charter members left.

A business session on Wednesday afternoon brought the three-day program to a close.

CLIFF. ACKLEY, *Secretary.*

VERMONT VETERINARY MEDICAL ASSOCIATION

The twenty-fifth annual meeting of the Vermont Veterinary Medical Association was held at the Brandon Inn, Brandon, July 13-14, 1933, with 25 members in attendance.

All speakers and clinicians on the program were members of the Association. Several papers on mastitis were read and thoroughly discussed. An all-day clinic was held under the direction of Dr. Herman Philipsen, of Brandon, with Dr. H. L. Mills, of Burlington, in charge of the small-animal clinic.

Mrs. Philipsen, assisted by other members of the Local Committee, entertained the ladies of the party at a luncheon at her home. This occasion was followed by a drive about the beautiful hills of Brandon and along the shore of Lake Dunmore.

More than fifty persons sat down to the annual banquet in the evening. Dr. L. H. Adams, of Montpelier, was toastmaster. Among the speakers was Mr. Conklin, secretary of the Ayrshire Breeders' Association, who strongly emphasized the fact that

the breeder should put his herd under the supervision of a graduate veterinarian. Miss Adele Miller, daughter of Dr. E. L. Miller, of Newport, ably demonstrated her ability as an artist of the dance. Little Dorothy Reed, of Newport, also entertained the gathering with acrobatic and dance numbers.

At the business session, one new member was added to the roster of the Association. The following officers were elected to serve during the coming year: President, Dr. N. H. Tenney, White River Junction; first vice-president, Dr. L. H. Adams, Montpelier; second vice-president, Dr. H. L. Mills, Burlington; secretary-treasurer, Dr. G. N. Welch (reëlected).

G. N. WELCH, *Secretary.*

NORTHWESTERN OHIO VETERINARY MEDICAL ASSOCIATION

The summer meeting of the Northwestern Ohio Veterinary Medical Association was held at the Hotel Harding, Marion, July 20, 1933, and was attended by about 125 veterinarians and their wives.

Dr. D. C. Hyde, Assistant State Veterinarian, of Columbus, presented a very excellent paper regarding Order No. 4 of the Division of Animal Industry, in which he referred to the present and past efforts of the Division to control the distribution of hog-cholera virus. Dr. J. R. Karr, of Coshocton, gave an interesting paper on the urine test for pregnancy. The afternoon session was in the form of a clinic under the direction of Dr. O. C. Alspach, of Marion, and Dr. Harry A. Hoopes, of La Rue.

WARREN P. S. HALL, *Secretary.*

MARYLAND STATE VETERINARY MEDICAL ASSOCIATION

The semi-annual meeting of the Maryland State Veterinary Medical Association was held at the University of Maryland, College Park, July 22-23, 1933, with about sixty in attendance.

The meeting was called to order by the President, Dr. Walter E. Campbell, of Bel Air. In his address, Dr. Campbell gave many worth while suggestions, based on his own experience, as to how the practicing veterinarian may adjust his costs to present-day conditions. These suggestions included a closer scrutiny of the costs of transportation, more care in the purchase of tried instruments and drugs, and the continuous watching of small finan-

cial leaks that, in the aggregate, greatly increase the costs of doing business.

Dr. Hulbert Young, of Baltimore, reviewed the life, work and contributions of the late Secretary of the Association, Dr. E. M. Pickens. Letters of condolence from prominent individuals both within and without the veterinary profession were read and incorporated in the minutes of the meeting.

Dr. R. A. Pearson, president of the University of Maryland, and Dr. H. J. Patterson, director of the Experiment Station and dean of the College of Agriculture, welcomed the Association to the University campus, and discussed briefly the relation of veterinarians to the live stock interests and the health of the human population.

A paper was presented by Dr. Mark Welsh, of the University of Maryland, summarizing the results of several years of work on the Maryland plan for the control of hog cholera. The results indicated that, under usual farm conditions in Maryland, cholera can be prevented by intelligent care in the feeding and management of the swine herd. Also, it was indicated that the use of virus must be restricted to the hands of competent veterinarians if this disease is to be curtailed.

Dr. J. N. Frost, of New York State Veterinary College, presented an instructive paper on "Diseases of Foals." This paper was particularly timely in view of the fact that there has been a marked increase in the colt population in many sections. Umbilical infections, colic, goiter, rupture of tendons, depraved appetite, rickets and other ailments were discussed from the practical viewpoint of control and cure. The technic and benefit of blood-transfusions from mother to young also were discussed, as well as the sanitary precautions necessary to prevent colt losses.

A short business session was held, at which poultry blood-testing work was discussed by Dr. T. A. Ladson, of Olney; Mr. J. B. George, director of the Maryland Live Stock Sanitary Service; Dr. C. M. Grubb and others. The Committee on Bovine Tuberculosis Testing, of which Dr. Ladson is the chairman, reported its findings, and the Association rescinded the action it had taken previously concerning charges for such testing.

By unanimous vote, the Association elected Mrs. E. M. Pickens an honorary life-member.

The afternoon was devoted to clinics. Dr. J. N. Frost conducted the large-animal clinic. Demonstrations of bleeding chickens for test purposes were conducted by Drs. H. M. DeVolt, F. W. Cruickshanks, John D. Gadd, and others.

Out of respect to the memory of Dr. Pickens, a banquet was not held.

The first paper on the second day, "Traumatic Indigestion and Its Surgical Treatment," was presented by Dr. C. M. Grubb, of Rockville. A brief discussion of the anatomy and physiology of the digestive organs of the ruminant preceded an interesting discussion of traumatic injuries to those parts. Drs. Grubb and L. J. Poelma gave an instructive description of the surgical removal of a wire puncturing the reticulum.

Dr. P. C. Underwood, of the Zoölogical Division, U. S. Department of Agriculture, Washington, D. C., presented a paper on "Heart-Worm Disease of Dogs." The paper dealt briefly with the rapid spread of the disease and discussed a treatment developed recently by the Zoölogical Division.

"The Control and Treatment of Gastro-Intestinal Parasites of Horses" was the title of a paper given by Dr. W. H. Wright, Zoölogical Division, U. S. Bureau of Animal Industry, Washington, D. C.

Major R. A. Kelser, V. C., U. S. A., Washington, D. C., presented a paper on "The Transmission of Equine Encephalomyelitis by Mosquitoes." An interesting discussion of the experimental transmission of the virus of equine encephalomyelitis by mosquitoes was presented with the aid of drawings and diagrams. Mosquitoes were permitted to feed on artificially inoculated guinea pigs and at varying times later were allowed to feed on susceptible guinea pigs. It was indicated that this disease is naturally transmitted by insect vectors, and that it is likely the mosquito plays a prominent part.

The small-animal clinics held in the afternoon were conducted by Dr. I. M. Cashell, of Washington, D. C. Several dogs affected with heart-worm disease were shown by Dr. Underwood, who also demonstrated the microscopic findings in this disease.

MARK WELSH, *Secretary.*

Virginia Women's Auxiliary Meets

The Women's Auxiliary to the Virginia State Veterinary Medical Association met at Fredericksburg, July 14-15, in conjunction with the meeting of the State Association. Twenty-five members and three visitors were in attendance. Entertainment included a luncheon and a tour of the many historic spots of the city. Officers elected for the coming year are: President, Mrs. R. E. Brookbank, and secretary, Mrs. A. J. Sipos, both of Richmond.

NECROLOGY

E. J. DECKER

Dr. E. J. Decker, of Far Rockaway, N. Y., died April 22, 1933. The cause of death was cerebral thrombosis. Born October 14, 1864, Dr. Decker was a graduate of the American Veterinary College, class of 1888. He joined the A. V. M. A. in 1893, and until his death was one of five members on the roll who joined the Association at the meeting held in Chicago in 1893. He is survived by his widow and one son.

PERCY GRAHAM

Dr. Percy Graham, of Red Springs, N. C., died suddenly of a heart attack, July 24, 1933.

Born at Penrith, Cumberland County, England, January 20, 1874, Dr. Graham spent the early years of his life in his native land. At the age of 19, he went to British Columbia where he worked on a ranch for a year. Returning to England, he was associated with a veterinarian in Bakewell for six years. Succeeding years saw him as a riding master in London and Edinburgh and as a hotel proprietor in Guernsey Isle. In 1912, he went to Cuba where he was connected with a ranch for a short while. Cuba was not to his liking, however, and he decided to go on to British Columbia. On his way, he stopped in Red Springs to visit the brother of a friend. The country and the inhabitants pleased him so much that he bought a farm and settled there.

Dr. Graham decided in 1915 to study veterinary medicine. Accordingly, he matriculated in the United States College of Veterinary Surgeons, at Washington, D. C., and was graduated in 1918. He returned to his new home at Red Springs and built up a splendid practice. He remained a British subject, however, and for several months during the World War was connected with the British Army, at Newport News, Va. Dr. Graham was a great

lover of animals, especially horses and dogs, and was an experienced horseman.

Dr. Graham joined the A. V. M. A. in 1925. He was also a member of the North Carolina State Veterinary Medical Association. He is survived by his widow (née Sue Powell), one daughter by a former marriage, and one sister. W. M.

HARMON E. NELSON

Dr. Harmon E. Nelson, of Mount Sterling, Ill., died at his home, July 28, 1933, after an illness which dated back about a year, and which compelled him to retire from active work several months ago.

Born in New Windsor, Ill., in May, 1890, Dr. Nelson attended local schools before entering the Ontario Veterinary College. He was graduated in 1912 and located in Mount Sterling shortly afterward. Surviving Dr. Nelson are his widow (née Etta Rippel), five sons, three sisters and five brothers.

CLAUDE S. CONNER

Dr. Claude S. Conner, of El Centro, Calif., died in Mercy Hospital, San Diego, Calif., July 29, 1933, the result of a skull fracture received in an automobile accident six days before. Dr. and Mrs. Conner and their son were returning to El Centro after a visit in San Diego. They were ascending a steep mountain about 70 miles out of San Diego, when a car coming down, and apparently out of control, smashed into them. Mrs. Conner had one knee fractured and the son had one leg broken.

Dr. Conner was a graduate of Kansas State College, class of 1909. E. F. K.

LEROY B. GRAHAM

Dr. LeRoy B. Graham, of Cedar Rapids, Iowa, died at his home, July 31, 1933, following an illness of several months.

Born at Brookfield, Mo., February 14, 1880, Dr. Graham spent the greater part of his life at Saint Joseph, Mo. He was a graduate of the Saint Joseph Veterinary College, class of 1909, and spent several years in the meat-inspection service of the U. S. Bureau of Animal Industry. He located in Cedar Rapids about twenty years ago and organized the Cedar Rapids Serum Company, of which he was president. In April, 1932, Dr. Graham was appointed City Dairy Inspector of Cedar Rapids.

Dr. Graham joined the A. V. M. A. in 1915. He was a member and former president of the South Side Commercial Club of Cedar Rapids. He is survived by one daughter and one sister.

SAMUEL T. PYPER

Dr. Samuel T. Pyper, of Cincinnati, Ohio, died at his home, August 10, 1933. He was executive secretary of the Ohio Valley Division of the National Dairy Council. He was 47 years of age.

Dr. Pyper was a graduate of Ohio State University, class of 1913, and formerly was food inspector for the Health Department of Dayton, Ohio, a position he held for 12 years. From 1926 to 1930, he was chief food inspector for the city of Cincinnati.

Dr. Pyper joined the A. V. M. A. in 1916. He was a member of the Ohio State Veterinary Medical Association, and was a member of Alpha Chapter of Alpha Psi Fraternity.

DAVID W. BURT

Dr. David W. Burt, of Battle Creek, Mich., died August 15, 1933, at the home of his brother near Battle Creek, where he had resided for the past year. Born at Simcoe, Ont., 79 years ago, Dr. Burt was a graduate of the Ontario Veterinary College, class of 1881, and had practiced at Buchanan, Mich., and Cornwell, Ill., until his retirement about a year before his death. He is survived by one brother and two sisters.

JOHN M. LICHTY

Dr. John M. Lichty, of Sioux City, Iowa, died in a local hospital on August 12, 1933, following a heart attack. He suffered a stroke about a week before his death while attending a national convention of the Fraternal Order of Eagles, in Cleveland, Ohio. He improved after a rest in Cleveland and returned to his home at Sioux City, but his condition became worse and he was taken to the hospital.

Born at Lancaster, Pa., October 12, 1868, Dr. Lichty received a common-school education. At the age of 18, he removed to Hartington, Neb., and in 1895, he moved to Yankton, S. Dak. Six years later he located in Sioux City, Iowa, and while there decided to take up the study of veterinary medicine. He entered

the Chicago Veterinary College and was graduated in 1910. He returned to Sioux City and entered practice there.

Dr. Lichty joined the A. V. M. A. in 1917. He was a member of the Iowa Veterinary Medical Association, a past president of the Interstate Veterinary Medical Association and a member of Gamma Chapter of Alpha Psi Fraternity. He was also a past president of the state aerie of Eagles. He was also an Elk. Surviving him are his widow, two daughters, one son, five brothers and one sister. G. P. S.

A. GIBSON

Dr. A. Gibson, of Birmingham, Ala., died suddenly, August 16, 1933, while on a professional call to a farm near Indian Springs. He was 64 years of age.

Following his graduation from the Ontario Veterinary College, in 1896, Dr. Gibson went to Calgary, Alberta, to manage a large ranch for Hon. Spring Rice. He served in this capacity for several years and then located in Birmingham which was a small mining town at that time. He built up a lucrative practice and was prominent in social and civic affairs.

Dr. Gibson joined the A. V. M. A. in 1897. He was a member of the Alabama Veterinary Medical Association. Surviving are a widow, one son and three daughters. D. A. P.

PERSONALS

MARRIAGE

Dr. W. W. Bailey (St. Jos. '14), of Sussex, N. J., to Miss Anne M. Hackett, of Middletown, N. Y., June 10, 1933, at Middletown, N. Y.

BIRTHS

To Dr. and Mrs. John Ramsey, of Fort Worth, Tex., a son, John David, June 13, 1933.

To Dr. and Mrs. Olof Norling-Christensen, of Chicago, Ill., a daughter, Gerda, July 30, 1933.

To Dr. and Mrs. J. Cevaal, of Oostburg, Wis., a daughter, Carol Jane, August 3, 1933.

PERSONALS

Dr. H. H. Adair (U. S. C. V. S. '08) has been appointed postmaster at Bristol, Va.

Dr. R. O. Suddath (A. P. I. '19) has removed from McRae, Ga., to Greensboro, Ga.

DR. M. E. EPPERSON (Ont. '33), of Barnard, Mo., has opened an office at Galion, Ohio.

DR. W. J. ANGERER (K. S. C. '33) has located at Atkinson, Ill., for general practice.

DR. W. E. PARKS (Corn. '19) has removed from Pine Plains, N. Y., to Millerton, same state.

DR. C. H. MILKS (Corn. '30), of Ithaca, N. Y., has entered general practice at Newark Valley, N. Y.

DR. C. C. SUNDSTROM (Colo. '30) reports a change of address from Los Angeles to South Gate, Calif.

DR. WALTER GUERKINK (K. S. C. '31), formerly located at Akron, Ohio, has removed to Baldwin, Wis.

DR. E. R. BRAUN (Wash. '29) has removed from San Anselmo, Calif., to Napa State Farm, Yountville, Calif.

DR. J. H. BOYD (Ont. '13), of Clayton, Mich., recently completed the construction of a hospital for small animals.

DR. HAROLD L. SMEAD (Corn. '32), formerly of Greenfield, Mass., is now located at 228 North 53rd St., Philadelphia, Pa.

DR. ARTHUR MAIKE (O. S. U. '33) has located at Bellevue, Ohio, taking over the practice of the late Dr. J. F. Blinzley.

DR. GEORGE KERNOHAN (K. S. C. '31) has requested a change of address from Palo Alto, Calif., to San Mateo, same state.

DR. F. C. STEINMAN (Tex. '29), formerly of Columbus, Ohio, now receives his mail at 5438 Kincaid Street, Pittsburgh, Pa.

DR. J. F. ROBB (Ont. '32), formerly associated with Dr. E. B. Cavell (Ont. '06), of Northville, Mich., has located at Saline, Mich.

DR. C. S. ELLIOTT (Ont. '84), of Greenville, Ohio, was retired from the service of the U. S. Bureau of Animal Industry, July 1.

DR. W. B. LINCOLN (Iowa '93), who has been in charge of federal meat inspection at Morristown, Tenn., was retired on August 1.

DR. D. H. DICKIE (Mich. '23), formerly with the Michigan Department of Agriculture, is now located at Sault Sainte Marie, Mich.

DR. WALTER G. GALLOWAY (O. S. U. '33) has located for general practice at Attica, Ind. He plans to operate a hospital in connection with his practice.

DR. C. B. MICHELS (Chi. '17), formerly of Chester, Ill., has accepted a position as meat inspector in the Saint Louis (Mo.) Department of Public Welfare.

DR. ANDREW SWANSON (Chi. '13) has removed from Atkinson, Ill., to Newhall, Iowa. He is suffering from polyarthritis and unable to practice at present.

DR. JAMES M. MILLER (McK. '13), of Benton Harbor, Mich., has been appointed Federal Farm Loan Agent. Dr. Miller was an unsuccessful candidate for sheriff at the 1932 election.

DR. GEORGE W. HESS (T. H. '18), of McHenry, Ill., has been appointed McHenry County Veterinarian by the Board of Supervisors, succeeding the late Dr. C. P. Draper (McK. '02).

DR. H. L. LYON (San Fran. '14), of Hillsville, Va., secured the Democratic nomination for a seat in the House of Delegates, at the Virginia primary elections held August 1. As such a nomination is practically equivalent to election, Virginia veterinarians look forward to having a member of the profession in the State Legislature at the next session.